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IRON DEFICIENCY ANAEMIA IN CAPE COLOURED AND AFRICAN CHILDREN IN CAPE TOWN*

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Routine haemoglobin estimations, carried out by one of us (P.L.) in the out-patient department of this hospital, showed that many Cape Coloured and African pre-schoolgoing children had low haemoglobin levels. Further haematological investigations (P.C.V., M.C.H.C., smear and bone-marrow, where indicated) showed that the low haemoglobin level was a manifestation of an iron-deficiency anaemia. Most of these children, however, on physical examination, had no evidence of anaemia. This observation led us to investigate the haemoglobin levels of apparently healthy children attending several crèches in the Cape Town area.

References to haemoglobin estimations on healthy children in South Africa are few.[1-5] None of the authors aimed at establishing a normal standard and, so far as we are aware, no standard of normality has been published for either European (White) or non-European (Coloured and African) children in this country. For the purpose of this investigation, therefore, Wintrobe's[6] standard of normal haemoglobin levels was accepted for comparison. Observations on a small number of White children from a high socio-economic group have shown haemoglobin levels almost identical to those of Wintrobe's normal standard.

MATERIAL

The children on whom haemoglobin estimations were carried out were all healthy Cape Coloured or African children between the ages of 1—7 years, attending local crèches. These probably represent a more privileged dietary group, since meals are provided at the crèches. Three municipal crèches for Coloured children, which were pooled, and one municipal crèche for African children were used in this investigation. In addition, 2 non-municipal crèches (Board of Aid and Windermere) for Coloured children were investigated.

The children at the 3 municipal crèches for Coloured children had been receiving 4 ml. (60 min.) Parrish's food (Syr. Ferr. Phos. Co.) daily for close on a year. At one non-municipal crèche (Windermere) iron tonics, which varied in nature and amount, had been given irregularly. The children at the remaining crèches had received no iron supplement to their diet.

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METHODS

All blood specimens were collected and haemoglobin estimations performed between the hours of 2 and 4 in the afternoon, and in the period September to December 1957.

Blood for haemoglobin and a smear was taken from the heel prick of infants and the thumb of older children, using a triangular cutting needle. Free, unrestricted flow without necessity of external pressure of any kind was obtained in all cases.

Capillary blood, 0·02 ml., was pipetted into 8 c.c. of 0·04% ammonia in water and the haemoglobin level read by the oxyhaemoglobin method using a Klett-Summerson colorimeter previously calibrated for the purpose against standard haemin and cyanmethaemoglobin solutions. A child was deemed to be anaemic if the haemoglobin was ½ g. below the lowest level accepted as the standard of normal for that age group.

RESULTS

Peripheral smears showed that many were normal, while others showed all gradations of an iron-deficiency pattern. As could be expected, the haemoglobin level was a far better index of anaemia than was the morphology of the red blood corpuscles on the smear.

Comparison of the mean haemoglobin levels of the various groups with the standard adopted (Figs. 1 and 2, and Tables I–IV) shows that the Cape Coloured and African children investigated have lower haemoglobin levels than normal. From each group those who fulfilled the criteria, outlined above, for anaemia were given iron therapy. By chance different iron preparations were available and were used in different groups as follows.

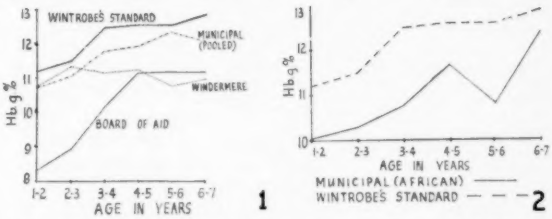

Fig. 1. Mean haemoglobin levels for Cape Coloured children at various crèches.

Fig. 2. Mean haemoglobin levels at a municipal crèche for African children (Langa).

TABLE I. MEAN HAEMOGLOBIN LEVELS. NON-MUNICIPAL CRÈCHE FOR CAPE COLOURED CHILDREN (BOARD OF AID)

Hb. g.%	Age in years 1-2	2-3	3-4	4-5	5-6	6-7	Total
5·5–6·5 ..	1	1	–	–	–	–	2
6·5–7·5 ..	–	1	1	–	–	–	2
7·5–8·5 ..	2	1	–	–	–	–	3
8·5–9·5 ..	2	3	1	–	–	–	6
9·5–10·5 ..	1	4	7	5	4	2	23
10·5–11·5 ..	–	2	6	8	2	3	21
11·5–12·5 ..	–	–	–	8	7	3	18
12·5–13·5 ..	–	–	–	1	1	–	2
Total cases ..	6	12	15	22	14	8	77
Average Hb. g.%	8·31	8·93	10·14	11·19	11·20	11·20	
St. deviation ..	±1·53	±1·48	±0·93	±0·87	±1·16	±0·83	
Wintrobe's standard g.%	11·2	11·5	12·5	12·6	12·6	12·9	

TABLE II. MEAN HAEMOGLOBIN LEVELS. NON-MUNICIPAL CRÈCHE FOR CAPE COLOURED CHILDREN (WINDERMERE)

Hb. g.%	Age in years 1-2	2-3	3-4	4-5	5-6	6-7	Total
8·5– 9·5 ..	–	–	2	–	1	–	3
9·5–10·5 ..	1	1	2	1	6	3	14
10·5–11·5 ..	2	4	8	7	5	1	27
11·5–12·5 ..	1	3	6	6	4	–	20
12·5–13·5 ..	–	–	–	–	1	2	3
13·5–14·5 ..	–	–	–	–	–	–	–
Total cases ..	4	8	18	14	17	6	67
Average Hb. g.%	10·78	11·35	11·19	11·28	10·80	11·00	
St. deviation ..	±0·92	±0·69	±0·91	±0·66	±1·00	±1·40	
Wintrobe's standard g.%	11·2	11·5	12·5	12·6	12·6	12·9	

TABLE III. MEAN HAEMOGLOBIN LEVELS. MUNICIPAL CRÈCHES (POOLED) FOR CAPE COLOURED CHILDREN

Hb. g.%	Age in years 1-2	2-3	3-4	4-5	5-6	6-7	Total
8·5– 9·5 ..	1	1	–	–	–	–	2
9·5–10·5 ..	3	2	1	–	2	–	8
10·5–11·5 ..	4	3	3	6	3	3	22
11·5–12·5 ..	3	1	9	21	14	5	53
12·5–13·5 ..	–	1	1	7	14	1	24
13·5–14·5 ..	–	1	1	–	5	2	9
Total cases ..	11	9	15	34	38	11	118
Average Hb. g.%	10·72	11·06	11·80	11·97	12·40	12·13	
St. deviation ..	±0·85	±1·31	±0·98	±0·61	±0·96	±0·94	
Wintrobe's standard g.%	11·2	11·5	12·5	12·6	12·6	12·9	

TABLE IV. MEAN HAEMOGLOBIN LEVELS. MUNICIPAL CRÈCHE FOR AFRICAN CHILDREN (LANGA)

Hb. g.%	Age in years 1-2	2-3	3-4	4-5	5-6	6-7	Total
5·5– 6·5 ..	1	–	–	–	–	–	1
6·5– 7·5 ..	–	–	–	–	–	–	–
7·5– 8·5 ..	2	2	–	–	–	–	4
8·5– 9·5 ..	2	5	1	1	1	–	10
9·5–10·5 ..	3	2	8	1	1	–	15
10·5–11·5 ..	2	1	7	3	–	–	13
11·5–12·5 ..	4	6	6	3	2	1	22
12·5–13·5 ..	1	1	–	1	–	–	3
13·5–14·5 ..	–	–	–	1	–	–	1
Total cases ..	15	17	22	10	4	1	69
Average Hb. g.%	10·02	10·30	10·77	11·66	10·80	12·40	
St. deviation ..	±1·84	±1·67	±0·96	±1·28	±0·51	—	
Wintrobe's standard g.%	11·2	11·5	12·5	12·6	12·6	12·9	

The children at the non-municipal crèche (Board of Aid) were treated with 0·28 g. (4·5 gr.) of ferrous gluconate (Ferlucon) in 3 divided doses daily for 24 days. The results of treatment, reflected in Fig. 3 and the accompanying Table V, show a statistically significant rise in the haemoglobin level of the various age groups, some of which were combined for statistical analysis because of paucity of numbers. The children at the non-municipal crèche (Windermere) were treated with 2 ml. (30 min.) of colloidal iron (Colliron) in 3 divided doses daily for 18 days. The results (Fig. 4 and Table VI) once again show a statistically significant rise in the haemoglobin level in various age groups.

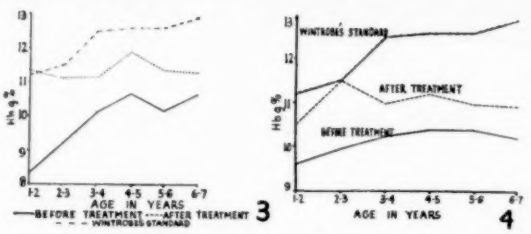

Fig. 3. Non-municipal crèche for Cape Coloured children (Board of Aid). Treatment with 0·28 g. (4·5 gr.) of ferrous gluconate (Ferlucon) daily for 24 days.
Fig. 4. Non-municipal crèche for Cape Coloured children (Windermere). Treatment with 2 ml. (30 min.) colloidal iron (Colliron) daily for 18 days.

TABLE V. NON-MUNICIPAL CRÈCHE FOR CAPE COLOURED CHILDREN (BOARD OF AID)

Age in years	Av. Hb. g.% before treatment	Av. Hb. g.% after treatment	No. of cases	Significance p
1–2	8·32	11·32	6	<0·001
2–3	9·22	11·13	9	<0·01
3–4	10·10	11·14	14	<0·002
4–5	10·65	11·90	12	<0·001
5–6	10·15	11·37	4	<0·002
6–7	10·62	11·30	5	
Total cases ..			50	

Treatment with 0·28 g. (4·5 gr.) ferrous gluconate (Ferlucon) daily for 24 days
All cases combined: $p < 0·001$ i.e. highly significant.

TABLE VI. NON-MUNICIPAL CRÈCHE FOR CAPE COLOURED CHILDREN (WINDERMERE)

Age in years	Av. Hb. g.% before treatment	Av. Hb. g.% after treatment	No. of cases	Significance p
1–2	9·60	10·50	1	
2–3	9·95	11·50	1	<0·01
3–4	10·22	10·99	5	
4–5	10·37	11·17	5	<0·05
5–6	10·37	10·96	8	
6–7	10·20	10·94	4	<0·02
Total cases ..			24	

Treatment with 2 ml. (30 min.) colloidal iron (Colliron) daily for 18 days.
All cases combined: $p < 0·001$ i.e. highly significant.

It was decided to treat the Coloured children in the pooled municipal crèches, who had been receiving the customary 4 ml. (60 min.) of Parrish's food daily for close on a year, with an increased dose, and twice the maximum B.P.C. dose 16 ml. (240 min.) was given in divided doses daily for 24 days. Repeat haemoglobin estimations at this stage did not show any significant change. Subsequently, Ferr. et Ammon. Cit. 1·35 g. (22·5 gr.) daily was given for 24 days and the haemoglobin levels showed a statistically significant rise (Fig. 5 and Table VII). The children at the municipal crèche for Africans (Langa) were given Ferr. et Ammon. Cit. in the same dosage and again a statistically significant rise in

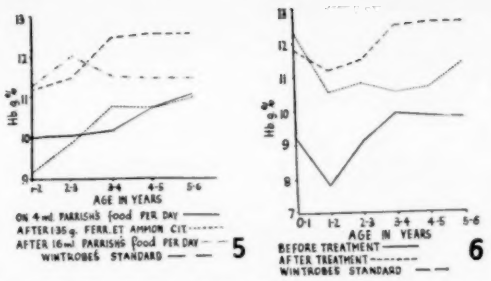

Fig. 5. Municipal crèches (pooled) for Cape Coloured children. Treatment with 16 ml. (240 min.) Parrish's food per day for 24 days followed by 1·35 g. (22·5 gr.) Ferr. et Ammon, Cit. per day for 24 days.
Fig. 6. Municipal crèche for African children (Langa). Treatment with 1·35 g. (22·5 gr.) Ferr. et Ammon. Cit. daily for 24 days.

haemoglobin levels in the various age groups was demonstrated (Fig. 6 and Table VIII).

13·8 g. of haemoglobin, found that the average haemoglobin in 132 Bantu school children on the rand was 97·48% and concluded that the average Bantu school child does not suffer from anaemia. Brock *et al.*,[3] in the Findings of the Cape Nutritional Survey, found 6·2% of Coloured school children and 3·1% of Bantu school children had a low level of haemoglobin. Le Riche[4] found that 2·5% of African children between 6 and 16 years on the Witwatersrand had a haemoglobin level below 12 g. and Kark and Le Riche,[5] using the 'Sicca' haemometer, found that the mean haemoglobin level of Bantu school children was 88·4% in boys and 87·75% in girls. In the present survey of pre-school-going children the incidence of anaemia was 64·9% for Coloured children and 66·7% for African children (Table IX).

TABLE IX. INCIDENCE OF ANAEMIA IN CAPE COLOURED AND AFRICAN CHILDREN

	Cape Coloured	*African*
Total Cases	262	69
No. Anaemic	170	46
Anaemic	64·9	66·7

TABLE VII. MUNICIPAL CRÈCHES (POOLED) FOR CAPE COLOURED CHILDREN

Treatment with 16 ml. (240 min.) Parrish's food daily for 24 days, followed by 1·35 g. (22·5 gr.) Ferr. et Ammon. Cit. daily for 24 days

Age in years	*Av. Hb. g.% on 4 ml. (60 min.) Parrish's food per day for 1 year*	*Av. Hb. g.% after 16 ml. (240 min.) Parrish's food per day for 24 days*	*No. of cases*	*Significance p*	*Av. Hb. g.% after 1·35 g. (22·5 gr.) Ferr. et Ammon. cit. daily for 24 days*	*No. of Cases*	*Significance p*
1–2	10·00	9·16	5		11·30	5	<0·01
2–3	10·08	9·90	4		12·03	4	<0·01
3–4	10·20	10·80	2	>0·05	11·55	2	
4–5	10·77	10·77	3		11·50	3	<0·02
5–6	11·10	11·03	8		11·51	7	
Total Cases ..			22			21	
All cases combined: p<0·05 i.e. not significant.					All cases combined: p<0·001 i.e. highly significant.		

TABLE VIII. MUNICIPAL CRÈCHE FOR AFRICAN CHILDREN (LANGA)

Age in years	*Av. Hb. g.% before treatment*	*Av. Hb. g.% after treatment*	*No. of cases*	*Significance p*
0–1	9·20	12·30	2	<0·001
1–2	7·83	10·58	4	
2–3	9·10	10·84	9	<0·001
3–4	9·94	10·59	7	
4–5	9·85	10·70	2	<0·01
5–6	9·80	11·40	1	
Total cases ..			25	

Treatment with 1·35 g. (22·5 gr.) Ferr. et Ammon. Cit. daily for 24 days.
All cases combined: p < 0·001 i.e. highly significant.

DISCUSSION

So far as we are aware no previous report has been published on the incidence of iron-deficiency anaemia in non-European children of the pre-school-going age in South Africa. The few references available deal with the incidence of iron deficiency anaemia in school-going children. Murray,[1] using the Tallqvist method, showed that the average haemoglobin in 543 poor-White children in a malarial area of the Northern Transvaal was 69·1%. Gear,[2] using a neoplasm haemometer in the scales of which 100% corresponds to

Several similar surveys in young children have been conducted elsewhere. Davidson *et al.*[7] showed that in the poorer classes of North East Scotland 41% of infants under 2 years and 32% of pre-school-going children were anaemic which they attributed to poor nutrition. Osgood and Baker,[8] in Portland, Oregon, found that in children between 4 and 13 years the haemoglobin ranged between 10 and 14 g. Colver[9] found that the average child under 3 years in South London was anaemic in relation to the 'iron standard', but that after 5 years the level lay in the limits of the 'iron standard'. MacKay[10, 11] established widespread existence of nutritional anaemia in breast-fed and bottle-fed infants in London.

From our results it would appear that iron deficiency is prevalent in pre-school-going Coloured and African children although it may not always be apparent. That this is not generally appreciated, is abundantly clear and we were, ourselves, surprised at the results. It is also evident that whereas most clinics, crèches and physicians prescribe vitamin preparations as a routine, iron therapy is far less widely prescribed although it is cheap and easily administered.

The importance of maintaining an optimal haemoglobin

level has been stressed by MacKay[12] who showed that routine iron medication in infants raised the resistance to infection and greatly reduced the morbidity rate. This is an especially important consideration in a non-European community where such factors as undernutrition, overcrowding and poor living conditions tend to increase the morbidity rate.

The object of treatment must be to provide iron in an available form and in adequate amounts to prevent or correct the deficiency. All the preparations used in this survey, with the exception of Parrish's food, were effective in raising the haemoglobin levels to a greater or lesser extent. It is to be clearly understood that no attempt was made to raise the haemoglobin to levels comparable with those of Wintrobe. The object of treatment was merely to demonstrate the children's ability to respond to iron therapy. For various reasons treatment was carried on for only a short while and it is obvious that the treated groups, although showing a significant rise, had not reached acceptable 'normal' levels. No untoward effects necessitating cessation of therapy were observed in any of the cases.

There are several practical points which are important to physicians in prescribing iron medication for children such as those in this study. The cost, bulk, time of administration, the availability of adjuvant substances, or the presence of antagonists, are the main ones. The briefest of comments is therefore offered along these lines.

Iron medication must be given in frequent doses since only a small percentage of any dose can be absorbed and small doses reduce gastro-intestinal tract disturbances. Iron should be administered between meals. McCance *et al.*[13] have shown that phosphorus-containing phytates and phosphates combine with iron to form an insoluble unabsorbable salt. Medicinal iron should, therefore, be given when the upper gastro-intestinal tract is relatively free of those materials. Milk and milk products contain large amounts of phosphates, and contrary to common belief, are not good vehicles for iron medication. The phosphate content in Parrish's food, apart from the relatively low iron content of this preparation, may, in our opinion, contribute to the poor results achieved with it.

Diets containing vitamin C greatly increase iron absorption through the capacity of the vitamin C to maintain iron in the reduced state.[14] This has been demonstrated by observations on absorption of iron from foods tagged with radioactive iron.[15] Citrus fruit juices are ideal vehicles for giving iron to infants because they not only disguise the metallic taste of iron, but their ascorbic acid enhances its absorption.[16]

Where iron-deficiency anaemia is the reason for therapy or where prophylactic iron is indicated, iron should be administered and compound haematinic preparations should be avoided. These latter are expensive, wasteful, irrational and confuse further blood studies.

SUMMARY

Haemoglobin levels of healthy pre-school-going Coloured and African children attending local crèches were estimated. Of the 262 Cape Coloured and 69 African children examined 170 and 46, respectively, were deemed to be anaemic.

This study was not intended to compare the efficiency of one preparation of iron with another. It does, however, show that an inexpensive and simple salt, Ferr. et Ammon. Cit., in a dose of 1·35 g. (22·5 gr.) daily, was an efficient haematinic for these children. Ferrous gluconate and colloidal iron were also effective, whereas Parrish's food, even in massive doses, was ineffective.

A plea for more extensive prophylactic and therapeutic iron therapy is made.

We are indebted to Prof. F. J. Ford, Prof. J. G. Thomson, Dr. C. Merskey and Dr. J. D. L. Hansen for their helpful criticism and advice, also to Mr. J. N. Darroch for help with the statistical analysis. We would like to thank Dr. C. Robertson for her co-operation and permission to use the municipal créches for these studies, the Board of Aid and Union of Jewish Women for permission to use their créches, Evan's laboratory for their supply of Ferlucon and Colliron, Miss D. Barker, social worker, Red Cross War Memorial Children's Hospital, for arranging visits to the créches, Mr. B. Todt for the photography and Miss D. Mendicoff for the illustrations and Mr. T. Norcott for technical assistance. We would also like to thank Dr. J. F. W. Mostert, Medical Superintendent, Red Cross War Memorial Children's Hospital, for permission to publish this work.

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THE WORLD MEDICAL ASSOCIATION

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Suid-Afrikaanse Tydskrif vir Geneeskunde : South African Medical Journal

VAN DIE REDAKSIE : EDITORIAL

YSTER

Yster en staal is vir die mens sedert die oertyd 'n simbool van sterkte en standvastigheid, soos die uitdrukking ‚'n man van staal' getuig. So vind ons ook in die Bybel verwysing na die eienskappe van yster: ‚en soos yster fynstamp en vergruis, sal hy dit alles fynstamp en vergruis'—Dan. 2 : 40.

Ystersoute word deur geneeshere gebruik sedert die dae van Hippokrates, maar selfs vandag is die finale uitspraak daaroor nog nie gelewer nie. Ysterterapie het waarskynlik uit die mistieke towerkuns en geneeskunde van die outyd ontstaan met die hoop dat 'n deel van die krag van yster oorgedra sou word aan die swak en sieklike pasiënt. Sydenham het, teen ongeveer die tyd van die volksplanting aan die Kaap, yster vir die behandeling van ‚chlorose van histerie' voorgestel. 'n Eeu later is yster in die as van bloed gevind en dit is vasgestel dat ysterbevattende voedsel die yster in die bloed laat styg. In 1832 het Pierre Blaud die spesifieke aksie van yster in die behandeling van chlorose bevestig en sy bekende, en ten regte, beroemde pille beskryf. Hierdie pille (0·3 g. ferrosulfaat en kaliumkarbonaat wat 0·1 g. ferrokarbonaat per pil beskikbaar stel) is vandag nog geregtig op 'n plek onder ons terapeutiese wapens. Sedert 1890 tot 1920 was yster tydelik in diskrediet weens dogmatiese stellings dat anorganiese yster sonder waarde in terapie is.[1] Die belangrikste rede was egter dat gevalle nie uitgesoek is nie en, soos vandag ook die geval is, is yster nie net spesifiek vir yster-tekortanemie toegedien nie, maar ook vir anemie as gevolg van ander oorsake. Die resultaat was natuurlik dat slegs die yster-tekortanemie gereageer het.[1]

In yster-tekortanemie sal yster, en yster alleen, die anemie herstel of ons nou die yster toedien met lewer per mond, bloedtransfusie of yster plus die so gewilde, maar so verdoemelike, breë spektrum hematiniese middels wat geen plek in die behandeling van anemie het nie.

Die effektiwiteit van yster is algemeen bekend in die behandeling van yster-tekortanemie (hipokroom, mikrosities). Die bivalente (Fe++) vorm is in die mens meer effektief as die trivalente (Fe+++) vorm, omdat yster in die bivalente vorm 1·5-15 maal beter absorbeer word as in die ander vorm. Honde, andersyds, toon egter nie hierdie verskil in absorpsie nie.

Ferrosulfaat of ferroglukonaat word toegedien en die gewone dosis is 3-6 tablette van 0·2 g. elk per dag. Blaud se pille moet in daaglikse hoeveelhede van 3-4 g. gegee word.

Daar is nie oortuigende bewys dat gemolibdeniseerde yster of verbindings van kobalt-yster meer effektief is as ferrosulfaat alleen nie.[2] Die dosis moet na etes toegedien word om die voorkoms van prikkeling van die maag teen te gaan. 'n Klein dosis ter aanvang, geleidelik vergroot, verminder die voorkoms van prikkeling van die maag en diaree. ‚Yster-enkefalopatie' en ileus na massiewe dosisse is gerapporteer[3,4] maar is uiters seldsaam. In kinders wat per ongeluk groot hoeveelhede ingekry het, is ernstige toksiese simptome waargeneem wat in tot 50% gevalle noodlottig was.[1]

Die parenterale toediening van yster was hoofsaaklik weens toksiese newe-effekte lank onsuksesvol. Vandag is die probleem oorkom deur ystersaccharaat te gebruik vir binnespierse toediening en 'n dextran-ysteroplossing vir intraveneuse- en binnespierse toediening.[1] In hierdie gevalle is dit wenslik om die presiese dosis yster te bepaal met die formule:[5] Yster in gram benodig = (normale hemoglobien—aanvanlike hemoglobien) × 0·255.

Twintig jaar gelede het Davidson en Fullerton[6] duidelik getoon dat ferrosulfaat ewe effektief was as ander ysterpreparate de tyds op die mark, maar teen 'n veel geringer koste.[6] In 'n onlangse artikel word die resultate van so 'n koste-vergelykende studie weereens gepubliseer.[7] In Skotland is gevind dat die koste 100 ferrosulfaat B.P.C. pille wat by 'n apteker gekoop is sonder 'n voorskrif, van 1/3 tot 7/4 gewissel het by 'n sekere handelsmerk van ferrosulfaatpille. Dit is gevind dat ferrosulfaat die goedkoopste ysterpreparaat is en dat dit net so effektief is as die duurder preparate. Ferrosulfaat behoort dus die eerste keuse te wees.

Davidson en Richmond[7] vind dat in die hospitaalpraktyk 46% van die ystervoorskrifte vir ferrosulfaat is en 54% vir die nuwer organiese ysterpreparate, terwyl in die algemene praktyk ferrosulfaat in 23% gevalle voorgeskryf is terwyl die duurder organiese preparate in 77% gevalle voorgeskryf is. Hulle bereken voorts dat as *slegs* ferrosulfaat voorgeskryf word, dit 'n besparing van £1,800 per jaar in die hospitaalpraktyk in Skotland sal beteken, terwyl £3,250 per *maand* in die algemene praktyk in Skotland op hierdie wyse bespaar kan word.

Klaarblyklik is daar die gevalle wat parenterale yster sal vereis en sekere ander verbindings beter sal verdra, maar waar die duur van behandeling dikwels oor 'n geruime tydperk strek, is die koste tog 'n faktor wat ons aandag vereis, veral as 'n middel wat 5-6 maal duurder is, nie meer effektief is as die goedkoper een nie.

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2. Neary, E. R. (1946): Amer. J. Med. Sci., **212**, 76.
3. Hurst, A. F. (1931): Guy's Hosp. Rep., **81**, 243.
4. Sjöberg, H. (1935): Acta med. scand., **85**, 129.
5. Brown, E. B., Moore, W., Reynafarje, C. en Smith, D. E. (1950): J. Amer. Med. Assoc., **144**, 1084.
6. Davidson, L. S. P. en Fullerton, H. W. (1938): Edinb. Med. J., **45**, 193.
7. Davidson, Sir. S. en Richmond, J. (1958): Lancet, **2**, 570.

INVESTIGATION OF HYPERSENSITIVITY

There are many approaches to the study of allergy. Some of these approaches involve the use of a single method or technique while others are based on a broad attack on the problem. Many new methods of investigation have been evolved in recent years and some of these have been considered in a report from the Mayo Clinic.[1] Although they

are of a laboratory nature these developments are of considerable clinical interest to the physician in practice.

Three procedures have become well established in providing data of inestimable value. These are the use of the ultracentrifuge which has made the precise determination of the molecular weights of protein molecules possible, the quantitative precipitin test which permits calculation of the ratios in which antigen and antibody molecules combine; and the studies on hapten—azoproteins which have helped to clarify the nature of the chemical groups which confer antigenicity and combine with antibody.

Electrophoresis has led to great advances in serological investigations and many modifications of the original Tiselius method have been used to study electrophoretic fractions for antibody activity. Albumin has never been found to have any antibody activity which resides essentially in gamma and beta globulins.

The technique of the gel diffusion has proved very useful. Test-tube studies (with the difficulty in obtaining precipitin reactions) can now be substituted by diffusion of antigen and antibody towards each other in agar gel; this results in complexes which form an opaque line or lines as each antigenic component in a mixture diffuses at its own rate. The technique of gel diffusion has been used in the study of pollen and dust extracts, for example, with material obtained from rabbits sensitized to grass pollens. Antigens from insect stings have been compared in a similar way and extensive studies have been made with this test on fractions of human serum in normal and diseased states.

Among haemaglutination tests the Coombs test and its variations have made extensive studies of human antibodies possible. These antibodies have been found in post-vaccinal encephalitis, blood dyscrasias, Guillain-Barré syndrome, glomerulonephritis, various collagen diseases and in several other conditions.

Using radioactive isotopes to tag antigen or antibody without significantly altering their immunological properties, globulins have been studied under various experimental conditions. Fluorescence has also been used as a method of tagging to demonstrate the localization of antigen and antibody in the tissues. The use of the technique of fluorescence has enabled certain workers to conclude that the plasma cell is a source of antibody. By other techniques involving cell transfer—isolating cells which have been exposed to antigen, injecting them into another animal of the same species, and demonstrating an increase in antibody titre—other workers have found the increase in antibody to be in proportion to the lymphocyte count. More work needs to be done to settle finally the question of whether plasma cells or lymphocytes are the site of antibody formation.

Many other procedures are available and newer ones will no doubt be forthcoming to supply the answers regarding the problems of antibodies and antibody formation. The studies on histamine are legion, and the action of 5-hydroxytryptamine and adrenocortical steroids are receiving much attention at present. The complicated problems of protein structure and synthesis, virology and genetics form the background for these investigations.

Clinicians should constantly keep themselves informed of the practical application of these intricate studies so that they may profit from the laboratory findings and suggest problems for further investigation.

1. Henderson, L. L. (1958): Proc. Mayo Clin., 33, 459.

AN UNPRECEDENTED CONTRIBUTION

The Southern Transvaal Branch of the Medical Association of South Africa recently made history by donating the unprecedented amount of £2,500 to the Benevolent Fund of the Association. By making this gesture this Branch has demonstrated to the whole of the medical profession what can be achieved by the enthusiasm and initiative of members. (A letter of appreciation from the Chairman of the Management Committee of the Benevolent Fund is published on page 44 of this issue.)

This contribution has a special significance in view of the fact that the financial position of the Association has, in recent months, been subjected to much criticism and scrutiny. It is not to be denied that the financial affairs of the Association have caused concern. Attempts made by the Association to procure greater support from its members, e.g. by means of special appeals, and the decision to increase the subscription, have, in some quarters, given rise to the criticism that the Medical Association is continually demanding more and more money from its members.

This inference is, to say the least, unjust. Like all public bodies the Medical Association has found that its expenses have increased considerably during recent years. It must, however, be remembered that the Association is not only continuing to render the usual services to its members, but that it has also greatly extended the range of its services. The services which are at present provided by the Association can be summarized as follows:

1. Opportunities for meeting colleagues, holding scientific meetings and providing a forum for the exchange of opinions.
2. A *Journal* for the spreading of medical knowledge.
3. Means for the settlement of ethical disputes between members.
4. Means for negotiating with medical aid societies and provision of some measure of control for medical benefit societies.
5. Means for negotiating with the Workmen's Compensation Commissioner.
6. Acting as the voice of the profession in all matters concerning medical practitioners, and being recognized as the official body in various Acts and Ordinances.
7. Legal protection for individual practitioners.
8. Procuring of income tax concessions of various kinds.
9. Obtaining preferential insurance of various forms for members.
10. Assistance to members by the Agency departments.
11. Amenities for members travelling overseas by reciprocity with the British Medical Association and the Canadian Medical Association, and through membership of the World Medical Association.
12. Improvement of salary scales of full-time personnel.
13. Influence on medical schools and medical education generally, e.g. the establishment of the College of Physicians, Surgeons and Gynaecologists of South Africa.

14. Postgraduate courses, provided directly or through medical schools.
15. Library facilities through grants to medical school libraries.
16. Assistance to needy dependants of members, through the Benevolent Fund.
17. Acting as a unifying factor, through Branches and Divisions, among practitioners.
18. Liaison with other professional bodies and the public.

Wholehearted support from all its members will make it possible for the Medical Association of South Africa to become a professional organization founded on unimpeachable ethical standards and imbued with the ideal not only to safeguard the financial status of its members, but also to give cultural guidance in matters of academic and professional policy and principles.

CONTROL OF ACID SECRETION IN THE SURGERY OF DUODENAL ULCER

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While the basic derangements responsible for peptic ulceration are not understood, the chronicity and recurrence of these ulcers appear to depend on the amount of acid that the stomach of the patient with gastric or duodenal ulcer produces. Treatment of a chronic peptic ulcer is therefore directed towards reducing the patient's acid secretion to a level where the ulcer will heal, and maintaining it at the level to prevent a recurrence of the ulcer itself or the development after operative treatment of a new one at the stoma.

The most effective and certain cure of an ulcer is bed rest; such treatment almost never fails to heal a benign lesion and its efficacy is based on regular frequent meals as a means of neutralizing acid as much as on mental and physical rest. An ulcer so treated heals 'as fast as epithelium can epithelialize'.[1] Unfortunately few people are able or willing, for economic reasons, to undertake a thorough regime of rest, and the history of most patients with chronic ulcer reveals repeated half-hearted attempts at such a cure over a number of years. Even a thoroughly supervised course of treatment which temporarily heals the ulcer is not always sufficient to protect the patient against a recurrence. Some patients are fortunate and attacks may not recur for a number of years; but where an individual becomes incapacitated every few months it becomes economically impossible to repeat further 4-6 week periods in bed.

The ambulant treatment of chronic peptic ulcer is often unsatisfactory, yet a certain proportion of patients remain well despite disregard of strict medical orders. Dietary regimes with milk as a mainstay, antacids and stopping smoking (and substituting food) help to neutralize the excess acid but do not reduce the amount secreted. Anticholinergic drugs are more successful in reducing acid experimentally than clinically.

There are few reports of psychotherapeutic cure of duodenal ulcer. Hurst's hypothesis of an ulcer diathesis and the relationship between stress and emotional factors to the genesis of the disease have been attractive theories, but there is little scientific support for such causal relationships. There is contradictory evidence in the literature concerning the influence of emotional and unhappy childhood experiences in the development of duodenal ulcer, although it is common experience that emotional crises do cause exacerbation of ulcer symptoms. On the known facts the development of an ulcer and excess secretion of acid are more closely allied to irregular eating habits, dietary deficiency or excess, and constitutional factors. These undoubtedly play a major role in the pathogenesis of the lesion. No social class is exempt; lorry drivers and business executives, labourers and doctors are alike affected. While all these patients are common prey to frustration and emotional tension, a more direct and clear insult to a stomach bathed in continuous secretion of acid is the occasional snack or quick cup of tea taken at irregular intervals. The high incidence of ulcer in the rural South Nigerian[2] and the peasant of South India, and the contrast between the frequent ulcer occurrence among Europeans and Indians in Natal compared with the African population, seem to indicate the importance of dietary factors. The increasing incidence among the latter cannot be explained simply as due to the increasing tensions of city living but is probably as closely related to changes in diet and habits of eating.

Constitutional factors have recently received emphasis by the demonstration of a significantly higher proportion of duodenal ulcer patients with group 'O' blood types than is found in normal control samples of the population;[3, 4] an equally striking demonstration of the familial trend is the evidence that there is a distinct pattern of duodenal ulcer in both patients and their relatives. A smilar correlation is found with gastric ulcer patients.[5]

The general aim of surgical treatment is to reduce the individual's acid-pepsin secretion to a low level. Surgical fashions have tempted the operator to apply a standard treatment to all patients with duodenal ulcer or gastric ulcer, with the exception of the very old and the very young. This has its advantages and disadvantages. On the one hand a surgical routine provides a technically better operative result; but some of the poor end-results of surgical treatment spring from the use of a single ablative technique for a disease with as wide a variation in degree and severity as such endocrine and autonomic disturbances as thyrotoxicosis and hypertension.

The proper aim of surgical treatment should be to reduce the acid-pepsin factor not according to a formula generally applied to all patients, but in proportion to the output of the individual patient. This places a considerable emphasis on the acid-secreting capacity of the stomach but, in the light of our present knowledge of the physiology of gastric secretion, it represents one measurable factor which can guide the extent and scope of operative treatment. It is therefore necessary to have a standard measurement of the acid output and a means of measuring it. There are at least 4 operations which will achieve a reduction in acid to a greater or lesser degree and the particular one chosen should be based on the acid output of the resting and active stomach, the length of history of the disease and the age and the sex of the patient.

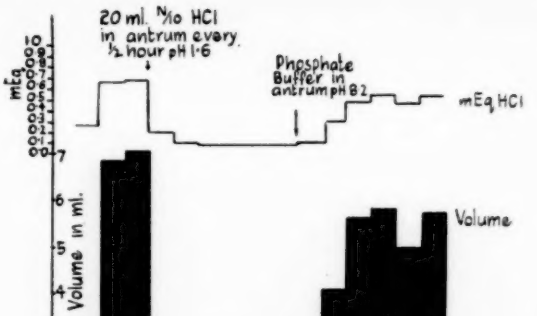

Fig. 1. Effect on acid secretion of acetylcholine in an exteriorized antrum pouch (Heidenhain pouch preparation). (The drawing at top applies also to Fig. 2.) *Fig.* 2. Effect on acid secretion of pH change in an exteriorized antrum pouch, indicating the inhibitory effect of acid in the antrum.

EXPERIMENTAL EVIDENCE

The experimental findings affording a rationale for the surgical reduction of acid production are based on Pavlov's concept of the 3 phases of gastric secretion:

1. *The cephalic phase*, mediated through the vagus nerve. Overwhelming evidence has been accumulated by Dragstedt[6-8] and his co-workers that in dogs abolition of this phase leads to a 60% or more reduction in the secretion of free hydrochloric acid. In man the quantity of acid reduction is not so predictable, since the human stomach is not dependent upon sudden outpourings of juice to deal with a large bolted meal, and secondly because night secretion (the interdigestive secretion) is not necessarily only vagal in origin. Yet marked reduction of the 12-hour night-secretion measurement following vagotomy is proof that this mechanism plays a predominent role in the hypersecretion found in duodenal ulcer patients. Three hundred duodenal ulcer patients with an average night secretion of 60 mEq. had, after vagotomy, an average free hydrochloric acid output of 5 mEq. in 12 hours. This reduction appears to be a permanent one.[7] An alternate mechanism for the hypersecretion of acid independent of the vagus nerve pathway has been suggested whereby stress phenomena cause the pituitary to secrete ACTH, liberating cortisone, which in turn stimulates an increase in acid and pepsin. Zubiran *et al.*[18,19] have shown that both ACTH and cortisone produce a sustained increase of free HCl in stomach pouches with and without vagal nerve supply. Since hypersecretion is abolished clinically and experimentally by vagotomy, it is clear that the excess secretory impulses are produced over the vagus nerves and not *via* the adrenal glands.

There is some experimental evidence that the vagus in fact plays a part in initiating the hormonal phase of secretion,[9] although other workers have failed to confirm this.[10] However, clinical data suggest that vagal and antral control of secretion forms part of an interrelated reflex arc and some of the value of removal of these nerves depends on this factor.[11]

2. *Gastric phase:* Gregory and Ivy[12] witnessed secretion from a stomach pouch transplanted to the submammary region when food was placed in the main stomach. Distension of the antrum was shown to produce secretion from such a transplant and procaine applied topically to the antrum prevented this response. This conclusively proved that the antral part of the stomach produces a hormone, 'gastrin', first postulated by Edkins in 1906.

Experimentally the stimuli for the production of 'gastrin' are food (particularly proteins), mechanical distension,[13] and acetylcholine. The latter affords presumptive evidence that cholinergic nerve endings are the trigger for the mechanism (Fig. 1). Dragstedt *et al.*[14] produced remarkable evidence of the power of the antrum to stimulate secretion when an antral pouch is transplanted to the colon, resulting in the application of constant *in vivo* distension stimulus.

On the other hand there is a growing body of evidence that the antrum not only produces a hormone which stimulates acid secretion, but that it acts as an inhibitor of acid as well.[15] Thus it is able to 'turn off the tap' of acid secretion in certain circumstances. Experimentally, inhibition of acid secretion from a Heidenhain pouch can be produced by a strongly acid solution placed in the exteriorized antrum (Fig. 2). Similarly the hypersecretion produced by an antral transplant to the colon is reduced if a portion of the antrum is left in its normal position exposed to an acid environment. There is no information of the relative importance in man of the antral and hormonal phases of secretion. The interrelation is too close for experimental separation; all that can be said is that in the experimental animal removal of each factor separately causes a profund drop in acid secretion.

3. *The intestinal phase* of secretion plays a relatively

small role and, despite earlier promising work with the hormone enterogastrone, little of clinical value has been achieved.

THE MEASUREMENT OF ACID OUTPUT

The strength of acid is measured by titrating a sample of gastric juice against 1/10 N NaOH. This gives the concentration of acid present. By measuring the total volume of gastric juice secreted in 12 hours, the final result is expressed as mEq/L of gastric juice. This provides a quantitative measurement of the output of acid.[7]

A multi-hole Levin tube is placed in the dependent part of the stomach and checked by screening to ensure that the tip lies in the antrum. Continuous suction with a low-pressure pump is maintained for 12 hours starting 4 hours after tea and a slice of toast, the patient being in a side ward or screened bed away from the sight and smell of food and with a sedative to ensure sleep. A sample is then titrated and the milli equivalents of acid estimated. This 12-hour secretion represents the acid output of a resting stomach.

For many years fractional test meals have been used and while they give a picture of the rise and fall of acidity and demonstrate definite differences between ulcer and normal patients, nevertheless in practice they are of little value in assessing the degree of acid production in an individual.

Histamine stimulation of secretion is more informative. Histamine is a specific stimulant of the gastric cells and there is evidence to support the view that it is identical with the hormone secreted by the stomach, if not the hormone itself. Unfortunately the size of the dose usually used, 0·5-1 mg., is not sufficient to produce more than a sudden sharp rise of acid secretion. Recently Kay[17] has shown that the side-effects produced by larger doses can all be overcome by an antihistaminic such as anthisan, which does not, however, affect the action of acid stimulation. This remarkable feature has no adequate explanation. Using this technique a true achlorhydria is only found in cases of pernicious anaemia.

It can be shown in experimental animals that repeated doses of histamine will produce a steady increase in acid output until the curve flattens out and the stomach is no longer able to produce more (Fig. 3). This supports Card's concept that the stomach can be stimulated to a maximum

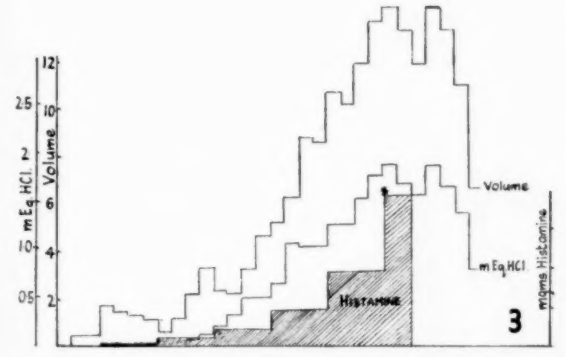

Fig. 3. Experiment showing the maximal output of acid by repeated and increasing doses of histamine (Heidenhain pouch).* The maximal acid output in this experiment was produced by 1·6 mg. of histamine.

capacity, and the rate and amount of secretion obtained is an index of the total number of parietal cells of the stomach.[18] Such tests on patients, using large doses in proportion to the patient's weight (0·04 mg./kg.) give an indication of the maximal total response of the stomach over three 20-minute periods and measured in mg. HCl or mEq represents the output of this 'parietal cell mass'.

The amount of acid secreted in 12 hours has been found, in a series of normal Indian and African patients, to be within a range of 5-20 mEq/L. This is in conformity with other workers' normal range with an upper limit of 20 mEq/L. Dragstedt[7] states that the average duodenal ulcer patient secretes 60 mEq/L, but analysis of a series of patients (to be published) shows that the range is wide, varying from just above the normal limit of 20 mEq/L to 100 or more mEq/L. It is reasonable to assume that a patient secreting 90 mEq/L in 12 hours, with a high level of secretion produced by maximal histamine stimulation and indicating a large total parietal cell mass, presents a different problem from one with 25 mEq/L output in 12 hours and a considerably smaller parietal cell mass that nevertheless responds to excessive stimuli by ulcer formation. An arbitrary dividing line at 60 mEq/L has been chosen between moderately high acid secretors and very high acid secretors. Such a distinction is felt to be of value in choosing an appropriate surgical procedure as indicated below.

SURGICAL APPROACH TO REDUCTION OF ACID

There are 4 approaches to the surgery of duodenal ulcer:

1. By-passing the ulcer by a gastro-enterostomy.
2. Wide surgical removal of acid secreting tissue by partial or subtotal gastrectomy.
3. Removal of the cephalic phase of secretion by vagotomy, together with drainage by gastro-enterostomy or pyloroplasty to obviate the mechanical side-effects.
4. Removal of the hormonal and cephalic phases of gastric secretion by antrectomy and vagotomy.

The removal of the acid secreting tissue is still the most widely used procedure. However, it is becoming increasingly apparent both experimentally and clinically that the effect produced by such a sledge-hammer approach, while usually meeting with 80-90% success, can in a majority of cases be at least rivalled by less radical procedures which attack the focal points of the pathological process of duodenal ulcer and with fewer resultant post-operative disabilities.

1. *Gastro-enterostomy*

Gastro-enterostomy was first performed in 1881 by Wolfler, using an anterior-long-loop antiperistaltic gastro-jejunostomy. This operation was modified by the use of a posterior loop and Petersen finally devised the posterior-no-loop procedure which Moynihan and Mayo advocated. Today there is generally a compromise between this and the original long-loop operation.

From 1900 to 1935 this procedure was the one of choice for duodenal ulcer. The operation apparently has little physiological basis and there is little if any reduction in acid output. In fact, experimentally it has been shown that there may under certain conditions be a rise in acid output.[46] In the 1920s, despite the continued popularity of the procedure amongst British and American surgeons, Finsterer and Eiselsberg in Europe had abandoned it in favour of partial

gastrectomy. By 1928 these surgeons were achieving mortality rates of under 3%.

There is little dispute that gastro-enterostomy will be followed by healing of almost every duodenal ulcer. The operation has however slowly fallen into disrepute as more and more evidence has accumulated that the rate of secondary ulceration is high. Sherren,[20] in the 1930s, is widely quoted as saying that non-recurrence after 2 years indicated a permanent cure. But the nature of this disease is such that recurrence in many cases only occurs after 2 or 3 years. The lack of a standard follow-up period is an unsatisfactory feature of most reports; these should be approached in the same way as malignancy reports by using definitive 5 or 10 year periods. It is the range in time of appearance of recurrence that explains some of the widely varying opinions of this operation.[23-29]

Patterson[21] in 1910 commented: 'The fear of recurrence casts a faint shadow over the otherwise admirable results of the operation' and he quoted a 2% recurrence rate. Bland-Sutton[22] was among the first in 1916 to indicate his objection by stating: 'With an unobstructed pylorus gastro-enterostomy cannot be relied on to cure the chronic duodenal ulcer and as it exposes it to regurgitant vomiting and jejunal ulcer I have gradually abandoned it as a routine method'. Yet Moynihan in his 12-year study gave his recurrence rate as 1·8%. The results of these surgeons in the early years of this century indicate how effective and flattering the immediate results sometimes are[25, 26] (Table I). But few patients in these years were consistently re-examined by X-ray and many of the post-operative symptoms, such as bleeding and dyspepsia, arouse the suspicion that some of these clinical syndromes were in fact recurrences.

TABLE I. RECURRENT ULCER FOLLOWING GASTROJEJUNOSTOMY

Author	*Year*	*No. of cases*	*Follow-up (years)*	*Recurrent Ulcers*		
				% Proven ulcers	*% Clinical ulcers*	*Total*
Lewisohn	1925	136	5—9	34	20	54
Luff	1929	744	6	2·8	6	8·8
Walton	1934	893	—	3·2	—	3·2
Wright	1935	17—30	5	4	4	8
Ogilvie	1935	—	—	20	—	15—20
Priestley	1948	—	—	—	—	15
Lahey	1945	—	—	—	—	15—20

TABLE II. TIME OF RECURRENCE OF ULCER SYMPTOMS AFTER OPERATION

Author	*Year*	*No. of cases*	*Symptom-free Interval (years)*	*Interval from 1st to 2nd operation (years)*	*Original operation*
Edwards *et al.*	1956	18	5·2 2·2	8 3·9	Gastro-enterostomy Gastric resection
Walters *et al.*	1955	301		11·2 3·7	Gastro-enterostomy Gastric resection
Everson *et al.*	1955	48	2 1	2 1	Gastro-enterostomy Gastric resection
Priestley *et al.*	1948	244	3·7 1·7	66% 10 yrs. later. 70% less than 5 yrs.	Gastro-enterostomy Gastric resection
Thompson	1956	63		60% 10 yrs. later. 66% in 5 yrs.	Gastro-enterostomy Gastric resection

A most interesting feature of these and other series, followed up over many years, is the comparison between the symptom-free interval following gastrojejunostomy and that following gastrectomy.[28, 30, 66-68] This symptom-free period is twice as long after the former operation and consequently the time of the second operation is delayed (Table II) for twice as long. Luff[24] in 1929 showed that of recurrent ulcers following gastrojejunostomy 68% delayed their appearance until a period of between 2 and 6 years had elapsed.

It will be seen that the rate of proven secondary ulceration ranges between 3-34% over large series of cases, many of which were followed up for 5 or more years. Priestley and Gibson,[28] reviewing the literature in 1948, state that the average incidence reported is 15%. Luff found a still higher recurrence rate following anterior gastrojejunostomy, the figure approaching 28% when an entero-anastomosis was added. Ogilvie's cynical view that the operation produces a patient 'who may be happy but is not safe' is a not unfair reflection.[27]

More recent results with a longer follow-up show clearly the trend towards late recurrence after gastrojejunostomy. Of a series of cases of proven gastrojejunal ulceration, the majority follow this operation. Marshall[31] found 77% of 172 cases followed gastrojejunostomy and 23% followed gastric resection. These figures, however, probably represent the end-result of many more gastrojejunostomies done in former years. The site of secondary ulceration is usually at the stoma or in the efferent jejunal loop (Thompson).[68]

2. *Gastric Resection*

Billroth laid the foundations for this type of operation. Other early surgeons concerned with this pioneer work were Rydygier in Poland (1881) and Braun (1892) who introduced afferent and efferent loop anastomosis. Kappeler in 1898 described a suspension method whereby the afferent limb is fixed above the stoma and the efferent limb into which the stomach empties—the forerunner of the Hofmeister valve.

The Billroth I operation was originally used for carcinoma and the anastomosis may be either end to end, with duodenum to greater curvature (Billroth-Finochietto), or with duodenum to lesser curvature (Horsley), or side of jejunum to stomach (von Haberer-Finnery).

Moloney[41] and Kanar *et al.*[38] recommend the Billroth I procedure for the following reasons: The operation is usually easier technically and the danger of leakage from a duodenal stump is removed. On the basis of the work showing that duodenal mucosa is more resistant to acid secretions than the jejunal mucosa, it is reasonable to assume that gastro-duodenal anastomosis is preferable to a gastrojejunal one. The nutritional state of the patients after the Billroth I procedure is considerably better, weight loss being present in only 10% as opposed to nearly 50% in Billroth II patients. The Billroth I operation presents fewer post-cibal symptoms and these tend to wear off with the passage of time. Serious nutritional deficiency following a Billroth II operation has been dealt with by a subsequent operation restoring gastro-duodenal continuity; Perman, quoted by Kanar,[38] reported 57 such successful conversions and Capper[34] states that 51 such operations from collected British sources were successful out of 60 cases.

Most authors agree that where less than 70% of stomach

is removed in duodenal ulceration there is a recurrence rate of nearly 10% (plus a 4% suspected recurrence) (Table III). With adequate high resection between 4 and 8%

TABLE III. RESULTS FOLLOWING BILLROTH I GASTRECTOMY

Author	*Year*	*No. of cases*	*Follow-up (years)*	*% Mortality*	*% Recurrence*
Horsley	1956	110	0–29	11	6
Walters *et al.*	1956	32	6–15	1·2	9
Capper *et al.*	1954	1000+			8·2<70% resection 0·9>70% resection
Goligher	1956	80	3–4	4	13·7
Wallensten	1954	159	4–20		7·5
Ordahl *et al.*	1955	35	3+		28·6
Kanar *et al.*	1956	248	1–8	3·1	3·5
Moore	1953	104	1–4	3·8	0
Harkins *et al.*	1954	266	1–6	2·6	1·3

develop stomal ulceration, a rate higher than that following the Billroth II operation.[32-40] This figure might still be the result of removing less than is excised in a Billroth II operation because of technical difficulty in bringing the cut end of the stomach to the duodenum.

The reduction of acid is said to approach anacid levels when 75% of the stomach is removed. Walters,[33] in 2 separate analyses, showed that approximately 40% of Billroth I patients developed achlorhydria, while 72-81% of Billroth II patients achieved this.

On the other hand, experimental work indicates that the Billroth I anastomosis is less likely to be followed by stomal ulceration than Billroth II operation. Kanar *et al.*,[38] in a series of experiments using hypersecreting gastric pouches after the method of Dragstedt, showed that there was a greater rise of acid secretion with the Billroth II anastomosis and that a larger number of animals died from stomal ulceration following Billroth II procedure.

The *Billroth II operation* is probably the most commonly used procedure today. The technique has become fairly standardized and many surgeons use the Hofmeister valve (Table IV). Unless 70% of the stomach is removed in cases

TABLE IV. RESULTS FOLLOWING BILLROTH II GASTRECTOMY

Author	*Year*	*No. of cases*	*Follow-up (years)*	*% Mortality*	*% Recurrence*
Walters *et al.*	1957	729	5–10	1·7	3·7
Wallensten	1954	322	4–20		2·5
Goligher	1956	106	3+	4·5	1·0
Ordahl	1955	64	3+	0·5	6
Moloney	1954	49			2
Moore *et al.*	1953	135	1–4	7·4	2·5
Thompson	1954	399	2–16	4·7	4·5

of duodenal ucler, the recurrent ulcer rate rises prohibitively.[34] When this is done it is generally agreed that the recurrence rate is distinctly lower than that following a Billroth I.[35-37,40-42,65] There is little to choose in the mortality figures, which range between 2 and 6%. Satisfactory results are obtained in 85-90% of patients and the operation has in this respect stood the test of 25 years practice. There is however a higher rate of minor, and sometimes disabling, digestive symptoms following a high subtotal gastrectomy, and persistent inability to gain weight is frequent.

3. *Vagotomy and Gastro-enterostomy*

This approach was first widely used by Dragstedt when in 1944 he introduced vagotomy alone. This soon proved an inadequate procedure as the end-results showed a high percentage of recurrent ulcers and many patients had a prolonged unpleasant convalescence. It was soon appreciated

TABLE V. RESULTS FOLLOWING VAGOTOMY AND GASTROENTEROSTOMY

Author	*Year*	*No. of cases*	*Follow-up (years)*	*% Mortality*	*% Recurrence*	*% Good results*
Tanner	1951	116	1–3½	0	3·6	88
Jordan	1952	460		1·4	6·0	93
Hoerr *et al.*	1953	147	2–4	0·5	6·0	90
Johnson *et al.*	1954	324	1–8	2	0	
Holt *et al.*	1954	243	1–7	0·5	1	97
Henson *et al.*	1955	89	2–3	1	12·4	
Dragstedt	1955	487	2–10	1·2	5·8	82
McEvedy	1955	184	3–6	1·1	1	94
Lloyd-Davies	1956	366	5–7	1·5	5	92·4
Crile *et al.*	1956	600	5–9	0·3	4·5	
McKelvie	1957	298	1–9	2·7	2	90
Hindmarsh	1957	197	2–8	0·5	2	90
Everson *et al.*	1957	178	3–11	1·1	7·6	82

that drainage of the stomach by a gastro-enterostomy overcame the unpleasant immediate side-effects and considerably lowered the recurrence rate. Thus vagotomy and gastro-enterostomy have now been widely practised for some 10 years and it is possible to begin to give an estimate of the end-results over the past decade (Table V).[48, 54-65]

Initially the objection was raised that the gastro-enterostomy in itself cured many ulcers and that doing this together with a vagotomy would not provide a fair reflection of the efficacy of vagotomy alone. This argument is a specious one, for the end-results of the procedure in terms of the patient's benefit is the proper criterion.

The reasons for the failure of the operation of vagotomy alone can be duplicated experimentally. A marked and continued hypersecretion of acid occurs after vagotomy in animals in which a pyloric obstruction has been produced, indicating that the excess secretion is of humoral origin.[43] Similarly vagotomy produces an increase in acid secretion from a Heidenhain pouch suggesting that distension, resulting from the reduced stomach mobility, stimulates acid secretion. This rise in secretion is effectively abolished by gastro-jejunostomy drainage.[44]

There are 2 aspects of the operation essential in providing a satisfactory result. Firstly, the vagotomy must be complete. This, to a large extent, is dependent on the experience of the operator. While the variations in the anatomy of the vagus nerves have been adequately described, in the vast majority of cases an anterior and posterior trunk are present and the latter is the more elusive. Unless these 2 large trunks are found beyond doubt the completeness of the operation is in question. Twigs must be looked for around the full circumference of the oesophagus and they are, when present, big enough to be obvious. Burge[45] has recently described an electrical aid to ensure the completeness of the vagotomy; he stresses that many vagotomies reported in the past were incomplete and this aid is an additional safeguard if required. There is an undoubted correlation between the number of recurrent ulcers and the number of incomplete vagotomies. With increasing experience of the operator the recurrences in a large series diminish to a level of between 2 and 6%. In 10 of 14 recurrent ulcers in an early series of 158 cases where vagotomy alone was used, there was a positive post-operative insulin response indicating incomplete vagotomy.[48] However, it should be stressed that when a post-operative insulin test is obtained this is not always the result of inadequate division

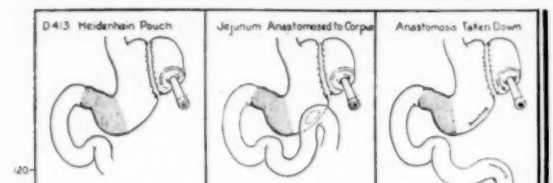

Fig. 4. The effect on acid secretion of gastrojejunostomy stoma in the fundus.

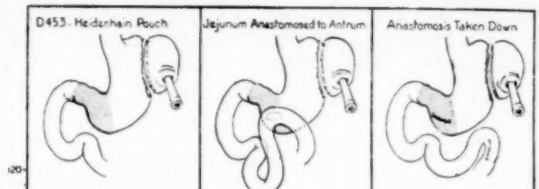

Fig. 5. The effect on acid secretion of gastrojejunostomy stoma in the antrum.

of the vagus nerves. A positive response following oesophagectomy has been obtained where there has been no doubt about the complete anatomical removal of all vagal nerve supply. Presumably some parasympathetic fibres are still carried by sympathetic fibres to the stomach.

Secondly, the site of the stoma is of importance. It has been well demonstrated that the stoma placed in the fundus of the stomach produces an increase in acid secretion.[46] This work has been extended and confirmed (Figs. 4-6*); the stoma should be placed in the antrum as close to the pylorus as possible.

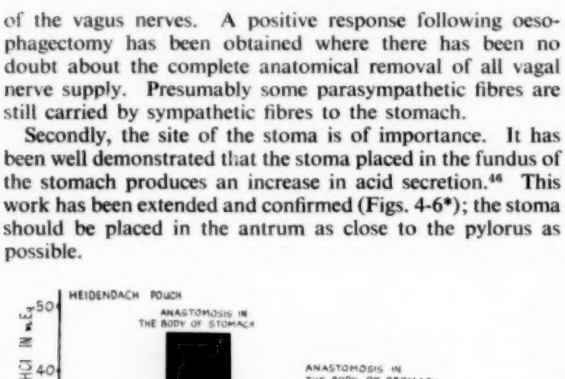

Fig. 6. The effect on acid secretion of varying the position of the stoma.

In 8 of 28 recurrent ulcers in a series of 487 cases, the stoma was found to be at the level of the mid-fundus, so allowing stasis in the antrum and producing hypersecretion.[48] It is of interest to note that in this later series the number of incomplete vagotomies fell to 11 in 400—3%.

Woodward[47] has recently shown that the size of the gastro-enterostomy stoma influences the level of acid secretion in dogs. When the size of the stoma was made larger than 4·5 cm. in diameter a threefold increase in acid secretion occurred, whereas small stomata produced no such increase.

Post-operative treatment following vagotomy and gastro-enterostomy is of importance, for there is always some degree of gastric paralysis. Gastric drainage by a Ryle's tube should be continued for at least 3-4 days and 12-hour gastric balances should be undertaken before finally dispensing with the tube. Perhaps the commonest post-operative complication of this operation is some degree of left basal congestion, and active breathing exercises and movement should be encouraged from the start.

A comparative series (Table V) shows that the operation of vagotomy and gastro-enterostomy takes its place as an effective surgical procedure in the treatment of duodenal ulcer. The mortality is under 1%, a significant reduction in comparison to any form of gastric resection. The recurrence rate is probably a little higher than large series of gastric resections indicate, but there are 3 further advantages which should be borne in mind: (*a*) The patient retains his stomach and therefore suffers less from dietary upsets, (*b*) weight loss is uncommon and considerably less than after any resection, (*c*) dumping and anaemia are seen less often.

A point of doubtful logic is frequently made—that one of the few indications for a vagotomy arises in cases of gastro-jejunal ulcer. If, therefore, the surgeon is wedded to high gastrectomy as the operation of choice, the omission of such a relatively straightforward additional manoeuvre as vagotomy seems to be contrary to the patient's best interests.

4. *Vagotomy and Antrectomy*

The hormonal mechanism of the antrum has been adequately confirmed experimentally. The hypersecretion of acid in animals due to a high-lying gastro-enterostomy stoma, or from a Heidenhain pouch after vagotomy of the main stomach, is completely abolished by subsequent removal of the antrum.[43,46] Clinical confirmation of the importance of this source of acid stimulus is seen in the observation that failure to remove all antral tissue in a partial gastrectomy is followed by a high rate of recurrent stomal ulceration. The Finsterer-Devine exclusion operation, with its prohibitive recurrent ulcer rate, was a clinical experiment testifying to this.

Waddell,[49] in a clinical study on patients having a 2-stage gastric resection, has ably demonstrated the active role of the residual antral tissue. He showed that the response of the remaining stomach to all stimuli (histamine, insulin, broth) is still present, although reduced because the antrum is

* Reproduced by permission of *Archives of Surgery*.[18]

excluded from the main food stream. Removal of the remaining antrum almost completely abolishes response to any stimuli. Waddell postulates that the reason for the low acid production from the stomach remnant, after gastrectomy with vagal fibres intact, is the interruption of a reflex mechanism. This consists normally of afferent impulses sent from the antrum which maintain the tonic state of the vagal reflex centres; a constant stream of efferent cholinergic impulses to the parietal cells is then carried by the vagus nerves. Interruption of either limb of this reflex by antrectomy or vagotomy diminishes the effect of the remaining source of acid stimulus.

Removal of the vagal and antral source may be obtained either by vagotomy and antrectomy or by vagotomy and antral exclusion. Both these are logical procedures based on physiological principles. Furthermore, they both preserve the main body of the stomach and in consequence avoid the physiological disturbances of digestion seen after removing wide areas of the stomach. These sequelae (weight loss, dumping, anaemia and inability to eat normally) are much less commonly seen when vagotomy is combined with antrectomy, antral exclusion or gastro-enterostomy than after removal of 70% of the stomach.

Edwards *et al.*[50] have followed 294 patients over a period of 1-10 years after vagotomy and antrectomy with a Billroth I or II reconstruction. No recurrent ulcer among these patients has so far occurred. Crile[51] and Farmer and Smithwick[52] report results more satisfactory than with gastric resection alone and Harkins, quoted by Edwards, in 68 cases over 4 years has had no recurrent ulceration.

The reconstitution following the limited 30-40% resection of the distal stomach lends itself to a gastroduodenal anastomosis. The method used in this department over the past 2 years has been the Horsley modification of the Billroth I, where the duodenum is joined to the lesser curvature of the stomach.[53] There must be few, if any, cases in which limited resection by such a technique is not possible.

There is in the minds of many surgeons considerable uneasiness at leaving a duodenal ulcer *in situ*. Extensive experience by many operators has shown that leaving such ulcers behind in difficult gastrectomies has very rarely led to any ill effect. Such excluded ulcers heal rapidly; vagotomy and gastro-enterostomy, by definition, leaves the original ulcer behind and the recurrent ulcer that may occur is a stomal one, not a recrudescence of the original ulcer.

Nevertheless, removal of the ulcer represents a technical danger, and anxiety over the duodenal stump after difficult dissections is a constant reminder of this. To obviate this danger Waddell *et al.*[49] has described the operation of vagotomy and antral exclusion which fulfils the physiological desiderata and excludes the risk of duodenal dissection. The distal half of the antrum is cut across and closed, a third to a half distal gastrectomy performed, and reconstitution is made by a Polya-Hofmeister anastomosis.

This operation retains the technical advantages of vagotomy and gastro-enterostomy and early follow-up reports indicate that the acid reduction and response to stimulation are satisfactorily depressed.

There are 2 clinical and experimental observations that are theoretically in conflict with the principle of this operation. The Devine exclusion procedure has fallen into disuse because of the high recurrence rate. The Waddell procedure differs in the addition of vagotomy and it remains for a long-term follow-up to see whether the reduction of the cephalic phase, and its interference with the hormonal phase, will permanently overcome the presence of retained and potentially functioning antral tissue. Secondly, experimental evidence is conclusive that an acid environment in the antrum inhibits acid secretion while an alkaline environment does not (Fig. 2). In the antral exclusion operation the antrum is constantly exposed to alkaline secretions from both the duodenum and its own mucosa. Harrison *et al.*,[15] on the basis of their experimental work, postulate that the antrum in an acid medium acts by producing an acid inhibitor rather than by simply stopping gastrin release. Whether the antrum in a permanently alkaline medium is of physiological significance is not yet known clinically. Woodward,[69] however, has shown that such an operation in dogs prepared with a Heidenhain pouch produces persistent hyperfunction, probably on the basis of a reflux of food and alkaline juices into the excluded antrum without the inhibitory effect of acid.

SUMMARY

The aim of surgical treatment of duodenal ulcer is to reduce the hypersecretion of the acid-pepsin factor of gastric juice; no operation, short of total gastrectomy, produces complete abolition. Successful surgical results depend not only on this low level of acid-pepsin reduction but also on a greatly diminished response to nervous stimulation *via* the vagus nerves.

The best means of assessing the activity of the stomach is the measurement of free HCl output under resting conditions for a period of 12 hours and under maximum stimulation by large doses of histamine. Using mEq. as the unit of measurement, it is possible to grade the level of acid output into high and very high secretors. The upper limit of normal acid output is 20 mEq/L; a figure of 20-60 mEq/L has been arbitrarily taken for a high secretor and above 60 mEq/L for a very high secretor.

The experimental work on the physiology of gastric secretion is reviewed; these observations have clarified the role of the vagus nerves in maintaining the hypersecretion of acid and the hormonal mechanism of the antrum of the stomach.

The differing surgical approaches to treatment of duodenal ulcer are reviewed and comparative figures in a series of cases from many sources over a long period of time are quoted. The reasons for the long-term failure of gastro-enterostomy as a definitive operation are indicated.

Wide removal of acid-secreting tissue by a Billroth II operation is a very successful procedure; but it is too radical an approach to apply to all grades of severity of this disease. The Billroth I operation is in some hands equally successful; but most authors show a higher recurrence rate, despite the less frequent occurrence of digestive and nutritional disturbances.

The operation of vagotomy and gastro-enterostomy is an established and successful procedure with a mortality rate significantly lower than that of partial gastrectomy and with less disturbances of digestion and nutrition resulting from it. Two essential features of the operation—complete section of the vagus nerves and the optimum position of the stoma in the antrum—are emphasized. Neglect of either of these aspects considerably increases the risk of stomal ulceration.

Extensive information has been gathered in the last 10 years for comparison of the relative merits of the two Billroth

operations and vagotomy and gastro-enterostomy, each of which interrupts part of a complex neurophysiological mechanism of acid secretion. A more fruitful approach is the application of the physiological principles of gastric secretion. Where the aim of an operation is marked reduction of acid secretion in terms both of quantity and of response to nervous stimuli, then vagotomy is obligatory as an adjunct to any such surgical procedure.

The problem of *which* suitable surgical procedure to adopt may be re-stated in terms of a choice between a vagotomy with a high gastrectomy, vagotomy with a low gastric resection (antrectomy), vagotomy with gastro-enterostomy, or vagotomy with antral exclusion.

The surgical ideal is to remove the ulcer, interrupt completely the vagal nerve supply and remove the antrum, leaving the patient with an amount of stomach adequate in capacity and digestive function.

Vagotomy and antrectomy appear to represent the closest approximation to this ideal surgical procedure for the majority of patients with moderately high acid secretion. For a very high acid secretor a vagotomy and high partial gastrectomy is indicated, with a Billroth I reconstitution by preference, or a Billroth II anastomosis.

Where extensive and difficult duodenal dissection is necessary a vagotomy and gastro-enterostomy is indicated, but with a very high secretor the difficulties of duodenal dissection must be weighed against the probability of recurrent stomal ulcer.

A vagotomy and gastro-enterostomy is indicated for young patients, particularly women, for thin patients who have been unable to put on weight for many years, and for elderly patients from whom a high morbidity rate is expected after major surgery.

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BESERING VAN 'N HIDRONEFROTIESE NIER

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Besering van 'n patologiese nier kom nie veel in die praktyk voor nie, en word selde in die literatuur aangetref. Daar is min leerboeke van chirurgie en patologie wat daarvan melding maak.

Gevalbeskrywing

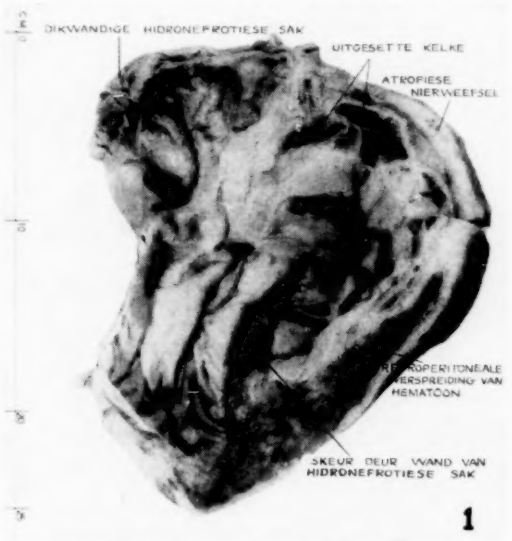
J.S., 'n naturelleman, 26 jaar oud, is op 10 September 1957 opgeneem met 'n matig-pynlike geswel in die buik. Sy geskiedenis was dat hy op 14 Augustus aan die linkerkant van sy buik geskop is. 'n Geswel het onmiddellik daarna ontstaan en dit was omtrent 5 dae lank pynlik. Daarna het die pyn bedaar hoewel die geswel voortbestaan het. Op 5 September het hy hom by die buitepasiënte-afdeling aangemeld; sy enigste klagte was die geswel in die buik. Hy het nooit naar gevoel nie, en het ook nie gebraak nie. Sy ontlasting was gereeld, en sy urinêre funksies is nooit vóór of ná die besering aangetas nie.

By ondersoek het ons 'n goedgevoede naturelleman gevind. Hart- en longondersoek het geen afwykings getoon nie. Die bloeddruk was 115/85 mm. Hg en die pols 70 slae per minuut en reëlmatig. Ondersoek van die buik het 'n groot geswel getoon wat vanuit die linkerlende en die linkerribrand, tot oor die middel-

lyn van die buik, gestrek het. Die geswel het kisteus en gespanne aangevoel, en was goed omskrewe behalwe waar dit onder die ribrand verdwyn het. Dit was nie gelobuleerd nie; die geswel was oral dof by beklopping, en die dofheid was nie aaneenlopend met die lewerdofheid nie. Dit was nie beweeglik nie. Die lewer en milt was nie vergroot nie, en rektale ondersoek het geen afwyking opgelewer nie. Die uriene was by roetiene-ondersoek normaal. Die diagnose van 'n traumatiese pankreaskiste is gemaak. 'n Bariummaal het getoon dat die maag na vorentoe en na regs verplaas was, soos gevind word met 'n pankreaskiste.

By operasie (19 September) deur 'n linker paramediane snit, is 'n groot retroperitoneale geswel gevind. Aspirasie van die geswel het donkerbruin vog gelewer wat soos veranderde bloed voorgekom het. Die geswel het vanuit die linkerlende gebult, en die maag en kolon was na voor en regs gedruk. Daar was geen verbinding tussen die geswel en die pankreas nie. Die afdalende kolon is gemobiliseer, en die geswel is van die mesenterium van die kolon losgedissekteer. Dit het geblyk dat die kiste nie van die linkernier onderskei kon word nie, en derhalwe is die kiste in sy geheel en saam met die linkernier verwyder. Die regternier het normaal gevoel.

Die monster (Afb. 1) bestaan uit 'n dikwandige hidronefrotiese



Afb. 1. Hidronefrotiese linkernierbekken met skeur in wand.

sak met 'n dun skil van nierweefsel oor die laterale oppervlakte uitgestrek. Die hidronefrotiese sak het 'n wigvormige skeur in sy inferolaterale deel gehad, en die skeur het in 'n geweldige groot kiste geopen. Die kiste het uit digte bindweefsel bestaan van die omliggende weefsel afkomstig. Die ureter was volledig afgesluit.

Histologies was die sakwand fibroties en xanthomateus as gevolg van 'n ontstekingsreaksie, en die nierweefsel het uit enkele uitgesette nierbuise bestaan. 'n Retrograde piëlogram, 2 weke na die operasie, het 'n normale regternierkelkstelsel aangetoon. 'n Maand na die operasie is die pasiënt in die buitepasiënte-afdeling ondersoek, en dit is gevind dat hy volkome herstel het.

BESPREKING

Dit is welbekend dat 'n besering van die buik met 'n stomp voorwerp tot 'n buikgeswel aanleiding kan gee. Die mees algemene geswelle wat ontstaan is 'n pseudokiste van die pankreas, of 'n mesenteriale hematoom. Dit gebeur soms dat na besering 'n bloeding binne 'n normale nierbekken—die sogenaamde ‚haematonefrose'—tot hidronefrose aanleiding kan gee,[1] maar dit is moeilik om te besluit of die intraluminale bloeding die oorsaak van die hidronefrotiese toestand is en of dit dié toestand gekompliseer het.

Die geweld nodig om 'n aangetaste nier—veral hidronefrose—te beseer, is gewoonlik van geringer aard as dié wat nodig is om 'n normale nier te beseer, en die besering kan ruptuur van die hidronefrotiese sak, of bloeding, veroorsaak. Die sak kan bars met uitstorting van die inhoud in die omliggende weefsels, en onderhewig aan die ligging van die skeur, kan dit intra- of ekstraperitoneaal geskied. In die eersgenoemde geval kan die sakinhoud (uriene, etter of bloed) 'n peritoneale prikkeling verwek en die klassieke beeld van peritonitis en skok soos by ruptuur van 'n buikorgaan word ook hier teëgekom. Wanneer ekstraperitoneale ruptuur voorkom, (in omtrent 75% van gevalle volgens Bailey[2]) is die beeld nie so uitgesproke nie. Verspreiding van die vog kan 'n retroperitoneale cellulitis verwek soortgelyk aan die toestand gevind by ekstraperitoneale skeur van die blaas. Skok mag voorkom, maar dit is waarskynlik van geringer aard, en 'n toksemiese beeld sal hier heers.

As bloeding na besering van hidronefrose voorkom, kan dit deur die wand beperk word en intraluminaal bly, of dit kan 'n ruptuur kompliseer met toename in die vog wat uitgestort word. 'n Massiewe bloeding sal dus 'n buikgeswel kan veroorsaak as dit beperk bly tot die retroperitoneale ruimte.

Baretz[3] beskryf 'n komplikasie van besering van hidronefrose wat hy 'n ‚renale hidrocele' noem—die sakinhoud versprei oor die nieroppervlakte deur 'n skeur deur die nierweefsel, of deur die bekkenwand naby die hilus. Die verspreiding word deur die nierkapsel-aanhegting beperk.

Spontane ruptuur van 'n hidronefrose is in die literatuur beskryf deur Reid en Menzies,[4] en Illingworth en Dick.[5] Waarskynlik vind spontane ruptuur as gevolg van geringe aanhoudende trauma plaas.

As gevolg van 'n skop in die linkerlende, het ons pasiënt 'n skeur deur die wand van 'n voorafgaande simtoomlose hidronefrose ontwikkel, en 'n massiewe bloeding daarvandaan het 'n hematoom in die naasliggende weefsels gevorm. Met die verloop van tyd het dit 'n dik fibreuse omhulsel gevorm deur die organisasie van die stolsel, en die kiste wat op dié wyse ontstaan het, het 'n verbinding met die hidronefrotiese sak gehad. Weens die ligging van die kiste, en die feit dat dit dwars oor die buik gelê het, is hierdie toestand klinies as 'n traumatiese pankreaskiste gediagnoseer. By operasie het die verwarring bly bestaan omdat die geaspireerde vog die kenmerke van vog afkomstig van 'n pseudopankreaskiste gehad het.

Ons wil dit hier stel dat die meganisme van die ontstaan van die toestand wat ons teëgekom het, dieselfde is as die meganisme van die ontwikkeling van 'n vals kiste van die pankreas—dat daar 'n hematoom in die retroperitoneale weefsels voorkom wat mettertyd 'n dik fibreuse wand daarom ontwikkel. Die kiste wat op dié wyse gevorm word bevat dus veranderde bloedelemente wat kenmerkend is van hierdie toestand.

OPSOMMING

1. Ruptuur van 'n hidronefrose, soos dit in die literatuur verskyn, en die differensiële diagnose van hidronefrose—veral as 'n geswel na besering verskyn, is bespreek.

2. 'n Geval is beskryf waar 'n simptoomlose hidronefrose met ontsteking aan 'n besering onderhewig was, met skeuring

deur die wand en retroperitoneale verspreiding van die kisteholte.

SUMMARY

1. Rupture of a hydronephrotic sac is discussed as it occurs in the literature, and the differential diagnosis of hydronephrosis is mentioned—especially when a mass appears after a traumatic incident.

2. A case is described in which a symptomless inflammatory hydronephrosis is subjected to an injury. A tear occurred through the thickness of the wall and haemorrhage within the sac caused a retroperitoneal spread of the cyst cavity.

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SALMONELLA AND SHIGELLA INFECTIONS IN AFRICA*

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A comparative survey of salmonellosis and shigellosis in Africa is at present impossible. It would require consideration of the clinical picture, including mortality, with regard to age, race, socio-economic structure, climate and locality; the morbidity should be ascertained and its influence on working capacity should be estimated; finally, it should give some information on the prophylactic measures which have been adopted in the prevention of the propagation of the infections.

Such comprehensive information is not available. During the last 40 years authors from different parts of Africa have reported on various aspects of salmonellosis and shigellosis; they have dealt with local findings. Pan-african reports on enteric fevers, as far as I know, have not yet been published. The vast dimensions of the task make this understandable. The very same reason forces me to consider the position in a small part of the continent, for which I have chosen the Union of South Africa. The review will be based on the fragmentary knowledge available in this country.

The relative importance of *S. typhi* infections as compared to the other salmonella organisms, makes it desirable to deal separately with the former and consider the rest under one heading.

The clinical picture of typhoid fever in South Africa seems to correspond to the classic description. The more severe cases are commonest among the non-Europeans, who seek medical attention at a more advanced stage. In most cases there is an excellent response to chloromycetin, but as a whole the prognosis is more serious in the non-European.

It is not known what proportion of the cases is diagnosed entirely on clinical grounds, in what proportion use has been made of the Widal reaction, and how often the diagnosis has been bacteriologically confirmed. The annual reports from the South African Institute for Medical Research[1] (SAIMR) reveal that in recent years about 8,000 Widal tests have been performed annually. If the prevalence in the rest of the Union were similar to that at the Witwatersrand, it might be estimated that the total number of Widal tests per year in South Africa would be 50,000-70,000; but the number of patients they represent is unknown. It emerges from the same reports, that *S. typhi* is isolated from about 400 specimens annually, which would correspond to 2,400-3,200 for the whole of the Union. Of these, 52% are from blood cultures. More than 90% of the strains isolated at the SAIMR ferment xylose. On antigenic analysis Lewin,[2] in 1937, found that 90·4% of the strains possessed both O- and Vi-antigens, that 7·9% were Vi-agglutinable and O-in-agglutinable and that 1·9% were devoid of the antigen Vi. Crocker,[3] in 1953, showed that approximately 65% of the strains belonged to phage type A and 10% to E_1 and that the types B_1, D_1, D_4, D_6, F_1, F_2, G, L_2, N, O, T and 28 occurred occasionally; 20% of the strains were not typable. Phage typing, ever since its introduction into the Union in 1942, has been centralized at the University of Pretoria. Although the services are free, many laboratories do not submit their strains regularly. This, of course, reduces the value of typing; but besides that it also appears that the epidemiologists of South Africa are reluctant to take advantage of the technique, so that presently it is merely of academic interest.

Typhoid fever is notifiable in South Africa. During the last two decades, according to official sources,[4] there have been approximately 4,000 cases annually in urban areas. This corresponds to an average notification rate of 25 per 100,000 population. The death rate is now about 1·88 per 100,000 and the case mortality varies between 3 and 7%. These figures are the best available, but they are considered to underrate the actual morbidity.[2] Firstly, no account has been taken of the rural districts; secondly, the reliability of the diagnoses is obscure; and, thirdly, the number of missed cases is unknown. Nevertheless, the figures indicate that the disease is fairly common and in spite of prophylactic measures the incidence has remained constant. This is contradicted neither by the increase in number of blood cultures at the SAIMR yielding a growth of *S. typhi* (1955, 114; 1956, 227; 1957, 258),[1] nor by the reduced number of *S. typhi* cultures submitted to phage typing during 1956 and 1957.[5]

We are still more ignorant of the carriers of *S. typhi*. It was estimated that 2% of the Bantu population were carriers.[6] The figure is disputable. All that is known is that 20,000 Vi-tests—immensely popular in tracing typhoid carriers—are performed annually at the SAIMR,[1] and 3-5% of them are positive in a dilution of 1 : 10 or more. The proportion of carriers among the reactors has not been established, but judged on theoretical speculations[7] it is probable that they constitute about 5% of the reactors, or 0·1% of the Bantu population.

Large-scale prophylactic inoculation of typhoid endotoxoid has been used in the prevention of the disease, but although there is no statistical proof of its effect, it is the general impression that it is beneficial.

It is usually maintained that salmonella infections other

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than typhoid fever are comparatively mild; but fatal cases have been described.[8, 9] In human pathology about 95% of the strains are recovered from excretions and 5% from blood and cerebrospinal fluid.[1] A recent survey at the SAIMR[10] showed that during 1957 we identified 59 different types; that *S. adelaide*, *S. labadi*, *S. typhi murium* and *S. london* were particularly frequent; and that 32 blood samples proved to contain salmonella, which were of 12 different types. On 2 occasions samples of cerebro spinal fluid yielded a growth of *S. typhi murium*.

As mentioned already, this type of infection is not notifiable, which renders the morbidity an unknown quantity. At the SAIMR[1] we have noticed a steady increase in the annual number of identified salmonella (1955, 244; 1956, 385; 1957, 515), but whether this reflects an increase in incidence or merely an expansion of bacteriological services remains uncertain.

The sources of salmonella infections in South Africa have been surveyed by Henning.[9, 11] He found that salmonellosis was very common among calves, but that cattle, horses, sheep and pigs were also infected. Moreover, he recorded salmonella epizootics among pigeons, canaries, ducks, turkeys and fowls, but not as yet among geese. Nor is there any reference to salmonellosis among fur-bearing animals, reptiles or arthropods. Neser *et al.*[8] recently reported a sample of biltong—salted, dried flesh of cattle or buck—to be heavily infected with *S. newport*.

Our ignorance of shigella infections is almost complete. As far as I know, from South Africa nothing has been published on their clinical aspects. Many shigella organisms are probably missed in the laboratories because of the duration of transport. Boardman *et al.*[12] in a recent survey on bacillary dysentery among African children found that 7% were infected with *Sh. sonnei* and 7% with *Sh. flexneri*. The latter was not classified in sub-groups. It is also my impression that the mentioned organisms are the most frequent, but at the SAIMR we frequently isolate *Sh. boydii*, occasionally *Sh. Schmitzii* and very exceptionally *Sh. shiga*. The latter is so rare that its existence in South Africa has been doubted.

Shigellosis is commoner in summer than in winter.[12] It is not notifiable *per se*; no information about the morbidity can be extracted from the reports of the Union Health Department, partly because all clinical dysentery, independent of aetiology, is classified together, and also because the non-European cases are not recorded. The annual number of identified shigella cultures at the SAIMR is increasing (1955, 169; 1956, 157; 1957, 259).[1]

It emerges from this review on enteric infections in Africa, and particularly in South Africa, that there are still many open questions. Much directed research is required before we can form a fairly accurate opinion on salmonellosis and shigellosis in Africa; in fact, it would be useful—and probably good economy too—if the health authorities appointed personnel with the sole duty of studying and combating these infections.

SUMMARY

Salmonellosis and shigellosis in South Africa are reviewed. Attention is drawn not only to our inadequate knowledge of the infective organisms and their distribution throughout Africa, but also to our ignorance of morbidity and mortality.

Notwithstanding the importance of individual observations as well as those of the health authorities, it is clear that much additional information is required. Some of it will undoubtedly be furnished by individual workers, but, for the bulk of the work, it will be necessary to call upon the health authorities.

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QUESTIONS ANSWERED : ANTWOORDE OP VRAE

IMMUNIZATION OF CHILDREN

Q—*Would you please suggest a scheme for immunization of children against diphtheria, whooping cough, tetanus, small pox and poliomyelitis with special reference to dosage, agents used, booster doses and possible contra-indications?*

A—In considering any scheme of immunization of children, the following facts must be borne in mind:

1. A passive immunity is transmitted to the new-born child by its mother against certain viral and bacterial diseases provided that the mother has suffered from them. Of special importance are poliomyelitis, measles and diphtheria.

2. This passive immunity begins to disappear after birth and is generally regarded as ineffective against severe infections after the age of 4 months, but may last for shorter or longer periods depending upon the degree of maternal immunity conferred at birth.

3. The production of antibodies by the new-born in response to active immunization is not effective, but increases steadily after the age of 1 month. Probably 3 months is the least age at which active immunization may be done.

The degree of immunity conferred by the mother must be related to her environment. In more highly civilized societies, where active immunization has been in force for many years, diseases tend to disappear, e.g. diphtheria. As a result, maternally-conferred immunity may be poor.

During their early years children are normally exposed to direct infections acquired from other children or carriers. Depending upon their previous state of immunity, these may act as primary or 'booster' infections.

In both these connections consideration must thus be given to the difference between crowded urban communities and sparsely inhabited rural areas.

Recent advances in antibiotic therapy enable treatment to be given to certain infectious diseases, e.g. chloramphenicol for pertussis. Diphtheria in the early months of life remains a very lethal disease. Neonatal tetanus carries a mortality rate of nearly 100%, but is a disease of primitive communities, and is not usually seen again until the child is walking. Poliomyelitis remains untreatable.

Ideally then, women of child-bearing age should be kept in a highly immune state against the diseases under discussion by inoculation at regular intervals. Since this is unlikely to be done, it is at least necessary that the proper courses of immunization should have been given at some stage, including injections for poliomyelitis.

Scheme for Inoculation

The following scheme is suggested for the inoculation of children to begin at the age of 5 months:

(1) A first injection of 'triple vaccine' of any reputable make. The dosage of the manufacturers should be adopted. 'Triple vaccine' immunizes against diphtheria, pertussis and tetanus, and should be given intramuscularly.

(2) Four weeks later, 1 ml. polio vaccine (S.A.I.M.R.) intramuscularly.

(3) Two weeks later, 2nd dose of 'triple vaccine'.

(4) Two weeks later, 2nd dose of 1 ml. polio vaccine.

(5) Two weeks later, vaccination against smallpox.

(6) Two weeks later, 3rd dose of 'triple vaccine'.

(7) Six months after the 2nd polio injection, give a 3rd dose.

N.B. All injections should be given with the child's head averted, so that the doctor and his instruments remain unseen.

As mentioned above, 'booster' doses are usually acquired by human contact, but it is customary to give a 'booster' dose of triple vaccine, and re-vaccinate shortly before the child goes to school, or at the age of 5 years, whichever is the earlier. The question of a 'booster' dose of poliomyelitis vaccine is still *sub judice*, but since it is such a widespread disease, and outbreaks of type I are so frequent, it would appear unnecessary in the general state of world health.

Contra-indications

Contra-indications are as follows:

1. *To vaccination*—eczema. In this disease, vaccination may usually be carried out about the age of 2 years, or if a smallpox epidemic occurs.

2. *To poliomyelitis vaccine*—possibly pregnancy. Penicillin-sensitive and highly allergic individuals always require special consideration. If the patient is known to be allergic to penicillin or other allergens, an antihistaminic (e.g. 2 mg. chlortrimeton) should be mixed with the vaccine and given at the same time. Local reactions would seem to be rare, but undoubtedly malaise, a mild meningo-encephalitis or even frank poliomyelitis may occasionally occur. Penicillin allergy is not an absolute contra-indication.

3. *To the 'triple vaccine'*—children not inoculated against poliomyelitis, in the presence of a severe epidemic of that disease. Local reactions may be severe, and malaise lasting 24-48 hours is not uncommon. If a reaction does occur at the first inoculation, it is advisable to mix an antihistaminic in the syringe when giving subsequent inoculations.

B.C.G. vaccination was not mentioned and is not discussed, but a good case can be made for giving this at birth.

THE ASSOCIATION'S FINANCES

The attention of members of the Association is drawn to the item printed in red on their subscription accounts directing their notice to the Hon. Treasurer's Special Fund. In amplification of this item the Hon. Treasurer has prepared the following memorandum:

Out of a total of 5,500 members only about 270 have so far responded to our appeal for donations. We are grateful for the amount of £1,200 which has so far been contributed and very much appreciate the kind remarks that accompanied most of the cheques. For the benefit of those members who have not yet contributed to our special fund, I am going to try once more to show how the money which members contribute in the form of subscriptions is being used—and why we must have more money to carry out our obligations towards members.

A. The *Journal*. It costs about £37,000 to produce the South African Medical Journal which is distributed free to every member. The *Journal*, however, just about pays for itself by means of advertising revenue, so that this item of expenditure need not be considered further.

B. The Medical Agencies (sale of practices, etc.). These services just about pay for themselves.

C. The rest of our income is derived from 2 sources: (1) Subscriptions to Head Office, thus far at the rate of £2 2s. 0d. a member, amounting to £11,300; (2) Commissions on insurances, amounting to about £4,000. The total income available for running the Association and its affairs, therefore, amounts to about £15,000.

Let us now see what we are expected to do with this £15,000:

Salaries to our Officials

The following is a list of our officials (excluding Editorial staff) and their remuneration.

	Per annum £
General Secretary (our chief administrative officer). Associate Secretary who deals with contract practice work. (A vast amount of work is done by this department). Assistant Secretary in Pretoria. (It is essential to have a liaison service with the Medical Council, W.C.A. Commissioner, Income Tax Commissioner and other Parliamentary bodies).	7,100
Business Manager who, in addition to other duties, is responsible for handling the advertisements in the *Journal* the revenue from which amounts to £37,000 a year	1,600
Typists, bookkeepers, secretaries to the 3 main Secretaries, addressograph machine operators, clerks, office boys, telephonists. (Every one of these people is indispensable in this big organization. People who do not know what the administration of the Association involves and who say we should prune our staff, are invited to acquaint themselves with the duties of every official)	6,700
Miscellaneous expenses	
Rent on floor space (Cape Town, Pretoria, Johannesburg) excluding editorial offices	1,520
Typewriter repairs, stationery, stamps, telephones, insurances	1,600
Delegates' expenses to the 2 Federal Council meetings (59 Federal Councillors), 2 meetings of Executive Committee, meetings of Central Committee for Contract Practice, travelling expenses for various members of Federal Council and our officials to special business meetings, commissions, etc.	4,600
Pension contributions	500
Contributions to medical libraries	800
TOTAL expenditure that has to be covered by income of £15,000	24,400

Fortunately, there were years when we were able to save money, and for the last 3 years we have drawn on previous investments to the amount of £21,000.

The reason for the increase in the expenditure is only partly accounted for by the increase in the salaries of some of our officials, by increased rentals and a few other items. The main reason for the rise in our expenditure is the decreasing buying power of the pound and the increased services rendered to our members. The pound is now worth about $\frac{1}{3}$ of what it was 15 years ago, so that subscriptions should really be raised from £2 2s. 0d. to £6 6s. 0d. In Great Britain subscriptions have gone up, so we learnt from Dr. Ian Grant recently, to 5½ guineas a year to the Head Office alone, and this despite a membership of 75,000. The point I really wish to make is that as far back as 1956 subscriptions should have been raised from £2 2s. 0d. to £4 4s. 0d. I am not going to repeat the unfortunate story of why this was not done at that stage.

Federal Council is not allowed to impose a compulsory levy, but we can ask members who can afford it to donate to their Association an amount equivalent or nearly equivalent to what they should have been charged over the last 3 years so that we can recoup investments on which we have had to draw.

It is an honour and a pleasure to work for our Association and to give up a considerable amount of time for that purpose. Some of the Federal Council members devote many days of their

time to this work. It is perhaps invidious to mention names, but I would like to pay tribute to people like Dr. Struthers, Dr. Sichel, the late Dr. J. S. du Toit, Dr. Maurice Shapiro, Dr. Grant-Whyte, Mr. Armitage, Dr. Waks, Dr. Schaffer, Dr. Turton, Dr. Leon Vercueil, Dr. Schneider, Dr. Lewis S. Robertson—and many others. Some of these people hardly ever take annual leave, but sacrifice their spare time for the benefit of our Association. Their burden will be greatly lessened if the financial position of the Association can be improved so that they need not worry unduly about necessary expenditure for services rendered.

AMPTELIKE AANKONDIGING

DIE BELEID VAN OPE LYSTE (OPEN PANEL)

Dr. L. M. Marchand, Medesekretaris, M.V.S.A., skryf: Geneeskundige dienste aan lede van bystandsverenigings en die vraagstuk van ‚geslote' of ‚ope' lyste is 'n onderwerp wat al heelwat bespreking in die Vereniging uitgelok het. Alhoewel ‚vrye keuse van dokter' nog altyd 'n aanvaarde beginsel was, het die Federale Raad eers in 1956 'n besluit oor die saak geneem. Moeilikhede het in sekere gevalle voorgekom met die uitvoering van die ‚ope lys'-beleid wat deur die Federale Raad aangeneem is, met die gevolg dat daar oorweging moes geskenk word aan omstandighede wat moontlik uitsonderings in die toepassing van die verklaarde beleid kon genoodsaak. Om hierdie rede het die Raad 'n memorandum, soos hieronder uiteengesit, as 'n leidraad vir Takke opgestel. Hierdie memorandum word vir inligting van lede van die Vereniging gepubliseer, sodat diegene wat in hierdie vorm van praktyk belangstel met die offisiële standpunt van die Vereniging bekend sal wees.

Om die posisie duidelik te stel, word die term ‚ope lys' gebruik vir die stelsel waarin enige geneesheer aan die diens mag deelneem en die lede van die bystandsvereniging toegelaat word om hul name op die lys van die dokter wat hulle verkies te plaas. In 'n ‚geslote lys'-stelsel word 'n bepaalde dokter aangestel om dienste te verskaf aan al die lede van 'n bystandsvereniging of aan 'n sekere aantal in 'n omskrewe gebied. Daar is vir die pasiënte geen vrye keuse van dokter nie.

In sowel die ope as geslote lys-stelsels ontvang die geneeshere 'n salaris wat bereken word volgens die hoofdelike gelde en die getal lede op hul respektiewelike lyste.

Die Memorandum

In April 1956 het die Federale Raad die volgende besluit aangeneem: ‚Dat die beleid van hierdie Vereniging sal wees om aan die pasiënt vrye keuse van dokter en aan die dokter vrye keuse van pasiënt te verseker. Ingevolge hierdie beleid behoort alle toekomstige aanstellings deur bystandsfondse op die basis van ope lyste vir algemene praktisyns en spesialiste te wees behalwe in buitengewone omstandighede.'

Sedert aanname van hierdie besluit het baie Takke groot moeilikheid ondervind om hierdie ‚ope lys-beleid' uit te voer want ‚buitengewone omstandighede' was nie omskrywe nie.

Toe die saak weer deur die Federale Raad in April 1958 behandel is, is daar moeilikheid ondervind om 'n definisie van ‚buitengewone omstandighede' te gee. Die gewone betekenis soos in die woordeboek aangegee (‚toestande wat ongewoon of nie gebruiklik is nie') sou nie noodwendig bevredigend wees nie, want verskillende Takke, waar toestande verskillend is, kon geneig wees om die saak van verskillende gesigspunte te beskou.

Om hierdie moeilikheid te bowe te kom het die Federale Raad die volgende besluit, wat nou van krag is, aangeneem:

> ‚Dat die beleid van die Vereniging in verband met die ope lys-stelsel sal wees om aan die pasiënt vrye keuse van dokter en aan die dokter vrye keuse van pasiënt te verseker. Ingevolge hierdie beleid behoort alle toekomstige aanstellings deur bystandsfondse en ander liggame op die basis van ope lyste vir algemene praktisyns en spesialiste gemaak te word. Onder die besef dat in die uitvoering van hierdie beleid daar praktiese moeilikhede mag voorkom, kan uitsonderings met die goedkeuring van die Federale Raad of sy Uitvoerende Komitee gemaak word.'

Met die uitvoering van die beleid wat deur die Federale Raad neergelê is, behoort Takke die volgende punte in gedagte te hou:

(1) Die posisie van geneeshere wat alreeds aanstellings by 'n fonds het, moet nie in gevaar gestel word nie. Alleen wanneer vakatures ontstaan of as nuwe poste geskep word, behoort die aanstellings ooreenkomstig die verklaarde beleid van die Vereniging in hersiening geneem te word.

(2) Alhoewel daar nog altyd 'n mate van teenstand teen die invoer van die ope lys-beleid was, selfs onder die geledere van die Federale Raad, het alle lede nog altyd saamgestem oor die beginsel wat daarin betrokke is, nl.: ‚Vrye keuse van dokter deur die pasiënt en van pasiënt deur die dokter.'

(3) Die redes vir die algemene aanvaarding van hierdie beginsel was dat alle raadslede oortuig was dat dit in die beste belang sou wees van

(*a*) die pasiënt wat onder hierdie omstandighede die beste moontlike mediese dienste sou kry, (*b*) die geneeskundige beroep, want alle dokters sou gelyke geleentheid hê om 'n bestaan te maak deurdat hulle toegelaat word om in die beskikbare werk te deel. Van hierdie 2 redes is die eerste natuurlik die vernaamste, want dit is tradisie dat die belange van die pasiënt bo alles anders gestel word.

(4) Op die laaste vergadering van die Federale Raad het dit geblyk dat raadslede baie meer besorgd was oor die beginsel wat daarmee verbonde is as oor die dringendheid van die onmiddellike toepassing van die beleid in daardie verenigings wat, om verskillende redes, daarteen was.

(5) Daar is op gewys, en dit is ook aanvaar, dat menige beginsel wat neergelê en aangeneem word nie onmiddellik uitgevoer kan word nie en dat die geleidelike invoering of die nie-onmiddellike toepassing van die beleid op 'n bepaalde vereniging nie die betrokke beginsel sou herroep nie.

(6) In die verlede was sommige Takke huiwerig om, buitengewone omstandighede' in verband met sekere verenigings te erken omdat hulle gevoel het dat hulle die beleid wat deur Federale Raad neergelê is getrou en onvoorwaardelik behoort te handhaaf. Dit het, by geleentheid, uitgeloop op 'n dooie punt in die onderhandelinge tussen 'n Tak en 'n vereniging, tot gevolglike nadeel van die betrokke pasiënte. Takke word dus nou daaraan herinner dat die Federale Raad nie begerig is om hom in die selfbestuur van Takke in verband met kontrakpraktyksake in te meng nie en is in werklikheid gretig dat enige onderhandelinge tussen 'n Tak en 'n bystandsfonds nie as gevolg van 'n Federale Raadsbesluit belemmer moet word nie.

(7) Derhalwe word alle Takke versoek om verdraagsaam in hulle houding te wees en om die ope lys-beleid op 'n redelike manier toe te pas; om te vermy dat betrokke verenigings in finansiële verleentheid beland wat moontlik aanleiding kan gee tot onderbreking in die geneeskundige dienste wat gelewer word aan die lede wat, per slot van rekening, die pasiënte is wie se belange altyd die swaarste moet weeg.

(8) Met ander woorde, as onmiddellike toepassing van die beleid, om ekonomiese of ander redes, op 'n bepaalde vereniging nie moontlik of prakties is nie, behoort Takke te streef na die geleidelike oopstelling van die lyste en die uitvoering van die beleid deur 'n proses van evolusie eerder as van revolusie. 'n Vereniging soos hierdie behoort aangemoedig te word om meer en meer poste te skep totdat die uiteindelike ideaal van 'n ope lys bereik word.

(9) Faktore wat 'n Tak moontlik kan beïnvloed om te besluit om vrystelling vir 'n bepaalde vereniging van die onmiddellike toepassing van die ope lys-beleid met betrekking tot aanstellings aan te beveel, is, onder andere, die volgende:

(*a*) Die inkomstegroep van die lede en die totale aantal lede wat aan die vereniging behoort.

(*b*) Die stand van die lede (dit wil sê hulle posisie op die maatskaplike leer).

(*c*) Die administrasiekoste en die moeilikhede waarmee die bystandsfonds met die invoering van die ope lys-beleid te kampe mag hê.

(*d*) Die beskikbaarheid van mediese personeel.

(*e*) Die moeilikhede wat sommige groot verenigings mag ondervind met die reëling van hospitalisasie en blokbesprekings van die operasiesaal.

OFFICIAL ANNOUNCEMENT : AMPTELIKE AANKONDIGING

MEDICAL AID SOCIETIES

SOCIETIES REMOVED FROM THE LIST DURING 1958

In order to assist members of the Association a list is given below of all the societies whose names were removed from the list of approved societies since January 1958. Members are again reminded that they should ensure that the name of a medical aid society appears in the list of approved societies before applying the preferential tariff to the members of such a society.

L. M. Marchand
Associate Secretary

Medical House
Cape Town
29 December 1958

Anglo-Alpha (Dudfield) Medical Benefit Society.
Caltex Medical Aid Society.
Hume Cape Medical Benefit Society.
Hume Transvaal Medical Benefit Society.
Hunt, Leuchars and Hepburn Ltd. (Durban) Employees' Medical Benefit Fund.

MEDIESE HULPVERENIGINGS

HULPVERENIGINGS VAN DIE LYS GESKRAP GEDURENDE 1958

Om lede van die Vereniging te help, volg hieronder 'n lys van die verenigings wie se name van die lys van goedgekeurde mediese hulpverenigings sedert Januarie 1958 geskrap is. Die aandag van lede word weer daarop gevestig dat hulle moet seker maak dat die naam van 'n mediese hulpvereniging op die lys van goedgekeurde verenigings verskyn alvorens hulle die voorkeurtarief op lede van sodanige verenigings toepas.

L. M. Marchand
Medesekretaris

Mediese Huis
Kaapstad
29 Desember 1958

Reunert and Lenz Ltd. Medical Aid Society.
S.A.A.M.E. Medical Aid Fund.
C. G. Smith and Co. Ltd. Medical Aid Fund.
Stuttafords Medical Aid Society.
2nd list:
Anglo-Alpha (Roodepoort) Medical Benefit Society.

THE BENEVOLENT FUND : DIE LIEFDADIGHEIDSFONDS

The following donations during November 1958 are gratefully acknowledged:

Votive Cards in Memory of:

Dr. T. A. Fuller *by* S.A. Society of Anaesthetists, Cape Western Branch; Mr. H. Hirschowitz *by* Dr. G. H. Bermann; Dr. G. Park Ross, Snr. *by* Dr. H. S. Gear; Dr. F. O. Fehrsen *by* Dr. H. S. Gear; Dr. J. S. du Toit *by* Dr. H. S. Gear.

Total Amount Received from Votive Cards £5 2s. 0d.

Services rendered to:
Dr. M. Byala *by* Drs. H. Grant-Whyte and partner, H. H. Navid, A. Radford, N. Shapiro, N. Walker and son, Mr. J. F. P. Mullins.
Dr. J. C. Faure *deur* Dr. P. E. Dreyer.

Met hartlike dank word die volgende skenkings gedurende die maand November 1958, erken:

The daughter of Dr. I. Kessel *by* Drs. S. S. Hersch and E. Lurie.
Kurt, son of Dr. F. J. Küpper *by* Dr. W. Emdin.
Dr. M. P. Royeppen and E. M. Gabriel, wife, and son of Dr. M. Gabriel *by* Drs. H. T. G. de Villiers, R. J. P. Venning, F. Walt and B. Murless.
Dr. W. Gilbert *by* Dr. H. Muller.

Total amount received for services rendered £41 13s. 6d.

Donations:	£	s.	d.
Legacy late Dr. R. H. Welsh	100	0	0
Cape Western Branch members collection box ..	2	13	3

Grand Total £149 8s. 9d.

PASSING EVENTS : IN DIE VERBYGAAN

Mr. Sidney J. Hersch, F.R.C.S., specialist surgeon, has moved from Clarendon Centre to 106 Lister Building, Jeppe Street, Johannesburg. Telephones: Rooms 22-3444, emergency 22-4191.

* * *

Dr. R. Sougin Mibashan, M.D., B.Sc. (Cape Town), M.R.C.P. (Lond.), M.R.C.P. (Edin.), is now in practice as a specialist physician at 1010 Medical Centre, Cape Town. Telephones: Rooms 3-3939, residence 44-5184.

* * *

Prof. L. J. te Groen, spesialis in verloskunde en vrouesiektes, wat ongesteld was, het weer herstel en hy sit sy praktyk voort te Mediese Sentrum 716, Pretoriusstraat 319, Pretoria. Die telefoonnommer van sy spreekkamers is 3-7242. Hy het nog geen huistelefoon nie, maar van die begin van Januarie 1959 af bly hy in die Koedoe Hotel.

* * *

Mr. A. J. Puttick, Ch.M. (Cape Town), specialist surgeon, wishes to inform his colleagues that he has commenced practice at Medical Centre, St. Matthew's Road, East London. Telephones: Rooms 3676, residence 8-8483.

Dr. A. J. Puttick, Ch.M. (Kaapstad), spesialis-chirurg, wil sy kollegas graag in kennis stel dat hy te Mediese Sentrum, St. Matthews-weg, Oos-Londen, begin praktiseer het. Telefone: Spreekkamers 3676, woning 8-8483.

* * *

University of the Witwatersrand, Medical Graduates Association. A meeting will take place on Friday 16 January 1959 in the Harveian Lecture Theatre, Medical School, Johannesburg, at 8.15 p.m. Prof. Emerson Day will speak on 'Early Detection of Cancer in General Practice'. Professor Day is the chief of the Division of Preventive Medicine at the Sloan-Kettering Institute, and Professor of Preventive Medicine in the Sloan-Kettering Division of the Cornel University College of Medicine, New York, USA. The Chairman of the meeting will be Dr. Lewis Robertson, Chairman of the National Cancer Association of South Africa. Professor Day's visit is sponsored by the *South African Practitioner*.

* * *

Dr. F. P. Reid, M.B., Ch.B. F.R.C.P., (Edin.), of Johannesburg, was elected a Fellow of the Royal College of Physicians of Edinburgh on 4 November 1958. Dr. Reid is a physician on the staff of the Johannesburg General Hospital and clinical tutor in the Department of Medicine of the University of the Witwatersrand. He is also honorary physician to the Bridgman Memorial Hospital.

Dr. F. P. Reid, M.B., Ch.B. F.R.C.P., (Edin.), van Johannesburg, is op 4 November 1958 gekies tot Genoot van die ,Royal College of Physicians of Edinburgh'. Dr. Reid is 'n internis op die personeel van die Algemene Hospitaal, Johannesburg, en kliniese studieleier in die Departement van Interne Geneeskunde van die Universiteit van die Witwatersrand. Hy is ook ere-internis aan die Bridgman-Gedenkhospitaal.

* * *

Beurse beskikbaar gestel deur Noristan Laboratoria (Edms.) Beperk. Vier beurse is beskikbaar gestel vir mediese studente in hulle finale jaar van die Universiteite van Kaapstad, Pretoria, Stellen-

bosch en die Witwatersrand om hulle te help om hulle studies te voltooi. Die waarde van die beurse is £120 elk en hulle sal jaarliks aan Unieburgers toegeken word deur 'n keurkomitee wat aangestel sal word deur die Raad van Direkteure van Noristan (Edms.) Beperk. Aansoeke moet skriftelik aan Noristan Laboratoria (Edms.) Beperk, Posbus 78, Silverton, Pretoria, gerig word deur die kantoor van die Dekaan van die Fakulteit van Medisyne van die betrokke Universiteit op 'n voorgeskrewe vorm op of voor 30 November van elke jaar. Vir hierdie jaar is die sluitingsdatum egter uitgestel tot 31 Januarie 1959. 'n Aanbeveling deur die Dekaan van die Fakulteit van Medisyne moet die aansoeke vergesel. Die keurkomitee sal nie slegs skolastiese bekwaamhede in aanmerking neem nie maar ook die geldelike behoeftes van die applikant.

* * *

The International Seminar for Public Health Workers for 1959, organized by the Central Council for Health Education, will be held from 21-24 April at the London School of Hygiene and Tropical Medicine, Keppel Street, London, W.C.1. The course is intended for medical officers, health educators, health inspectors, social workers and others concerned with the health education of the public. An introductory address will be given by Prof. Richard G. Bond, School of Public Health, University of Minnesota. The main emphasis of the programme will be placed on discussion meetings followed by lectures which will include a critique of these discussions. Practical instruction on the techniques of health education and the production of visual aid materials will also be given. The course will be non-residential, but accommodation in nearby hotels will be reserved for those who require it. The fee for attendance at the seminar will be approximately £6 6s. 0d. Applications for enrolment should be made to the Medical Director, Central Council for Health Education, Tavistock House, Tavistock Square, London, W.C.1.

* * *

The Fourth International Medico-Surgical Film Festival will be held at Pentecôte, Cannes, France, on 17-23 May 1959. The subjects for 1959 will be Neuropsychiatry and Neurosurgery and Mental Hygiene. Those practitioners interested in attending this Festival or in submitting a film to be shown at the Festival may obtain brochures and entry forms from this office or contact the Secretary of the Festival: Dr. Debay, Secrétariat, Festival International du Film Médico-Chirurgical, Palais des Festivals, La Croisette, Cannes, B.P. 279. Hotel accommodation, social events and sightseeing trips are being organized by the Committee of the Festival. There are 4 categories of films: (1) Teaching Medico-Surgical Film treating a freely chosen subject, (2) Teaching Medico-Surgical Film treating the imposed subject, (3) Health film treating a freely chosen subject, and (4) Health film treating the imposed subject. Each nation is allowed to enter only one film of each category, making a total of 4 films. These films are 'officially' authorized to enter the competition if they have been chosen by the official Authorities to represent the country concerned. When a Nation is thus officially represented, no other film forwarded to Cannes on 'private standing' can be accepted. However, if a Nation is not officially represented, one film of each category can still be allowed to enter the competition.

BOOKS RECEIVED : BOEKE ONTVANG

Diseases of the Nervous System. Described for Practitioners and Students. 9th Edition. By Sir Francis Walshe, M.D., D.Sc., F.R.S. With chapters on *The Neurological Complications of Liver Disease and Hepatolenticular Degeneration.* By J. M. Walshe, M.R.C.P. Pp. xvi+373. 60 Figures. 30s. net+2s. 6d. Postage Abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1958.

Die rhinogenen Erkrankungen der Orbita. Von Dr. R. Herrmann. viii+83 Seiten. 42 Abbildungen. DM 18.00. Stuttgart: Georg Thieme Verlag. 1958.

Coffee and Caffeine. By Dr. Med. Rolf Ulrich. Pp. 52. 7s. 6d. Bristol: John Wright & Sons Ltd. 1958.

Hearing Therapy for Children. Second, Revised Edition. By Alice Streng, M.A., Waring J. Fitch, M.A., LeRoy, D. Hedgecock, Ph.D., James W. Phillips, M.D. and James A. Carrell, Ph.D. Pp. 353. Illustrations. $6.75. New York and London: Grune & Stratton, Inc. 1958.

Principles of Research in Biology and Medicine. By Dwight J. Ingle, B.S., M.S., Ph.D. Pp. ix+123. 30s. net. London: Pitman Medical Publishing Co. Ltd. 1958.

Care of the Premature Infant. By Evelyn C. Lundeen, R.N. and Ralph H. Kunstadter, M.D., F.A.C.P., F.A.A.P. Pp. xv+367. 87 Illustrations. 60s. net. London: Pitman Medical Publishing Co. Ltd. 1958.

Progress in Radiation Therapy. Edited by Franz Buschke, M.D., with 13 contributors. Pp. iii+284. Illustrations. $9.75. New York and London: Grune & Stratton, Inc. 1958.

Physico-Chimie Biologique et Médicale. By Christian Bénézech. Pp. vii+684. 235 Figures. 8.000 fr. Paris, Masson et Cie. 1958.

Rehabilitation in Industry. Edited by Donald A. Covalt, M.D. Pp. xi+154. Illustrations. $6.00. New York and London: Grune & Stratton, Inc. 1958.

Muir's Text-book of Pathology. 7th Edition. Revised by D. F. Cappell, C.B.E., M.D., F.R.F.P.S., M.R.C.P., F.R.S.Ed. Pp. xx+1201. 733 Figures. 70s. net. London: Edward Arnold (Publishers) Ltd. 1958.

Basic Surgery. Edited by Leslie Oliver, M.B., B.S. (Lond.), F.R.C.S. Pp. xvi+1360. 680 Illustrations (including 4 coloured plates). £6 6s. 0d. net. London: H. K. Lewis & Co. Ltd. 1958.

Obstetrical Practice. 7th Edition. By Alfred C. Beck, M.D. and Alexander H. Rosenthal, M.D. Pp. xiii+1115. Illustrations. $14.00. Baltimore: The Williams and Wilkins Company. 1958.

Human Parturition—Normal and Abnormal Labor. By Norman F. Miller, B.S., M.D., F.A.C.S., A.C.O.G., T. N. Evans, A.B., M.D., F.A.C.S., A.C.O.G. and R. L. Haas, A.B., M.D., F.A.C.S., A.C.O.G. Pp. 248. 66 Illustrations. 60s. London: Baillière, Tindall and Cox Ltd. 1958.

Operative Surgery. Volume 4. *Head and Neck and Clearance of Lymph Nodes, Vascular Surgery, Endocrine Glands.* Under the General Editorship of Charles Rob, M.C., M.Chir., F.R.C.S. and Rodney Smith, M.S., F.R.C.S. Pp. xiii+163 (Part VI)+144 (Part VII)+66 (Part VIII)+4 (Index). 631 Illustrations. (This work consists of 8 Volumes at £5 10s. 0d. for each volume and an Index at £2 0s. 0d.). London: Butterworth & Co. (Publishers) Ltd. South African Office: Butterworth & Co. (Africa) Ltd., P.O. Box 792, Durban. 1957.

Operative Surgery. Volume 5. *Orthopaedic Surgery.* Under the General Editorship of Charles Rob, M.C., M.Chir., F.R.C.S. and Rodney Smith, M.S., F.R.C.S. Pp. xiii+366 (Part IX)+ 5 (Index). 584 Illustrations.

Operative Surgery. Volume 6. *Hand, Amputations, Plastic Surgery, Gynaecology and Obstetrics.* Under the General Editorship of Charles Rob, M.C., M.Chir., F.R.C.S. and Rodney Smith, M.S., F.R.C.S. Pp. xiii+105 (Part X)+71 (Part XI)+131 (Part XII)+123 (Part XIII)+4 (Index). 697 Illustrations.

Operative Surgery. Volume 7. *Breast, Genito-Urinary System* Under the General Editorship of Charles Rob, M.C., M.Chir., F.R.C.S. and Rodney Smith, M.S., F.R.C.S. Pp. xi+43 Part XIV)+273 (Part XV)+3 (Index). 497 Illustrations.

Operative Surgery. Volume 8. *Neurosurgery, Eyes, Ear, Nose and Throat.* Under the General Editorship of Charles Rob, M.C., M.Chir., F.R.C.S. and Rodney Smith, M.S., F.R.C.S. Pp. xv+57 (Part XVI)+83 (Part XVII)+196 (Part XVIII)+3 (Index). 534 Illustrations.

Disinfection and Sterilization. By G. Sykes, M.Sc. (Lond.), F.R.I.C. Pp. xviii+396. 17 Figures. 7 Plates. 75s. net. London: E. & F. N. Spon Ltd. 1958.

Skin Grafting. 3rd Edition. By James Barrett Brown, M.D. and Frank McDowell, M.D. Pp. xv+411. 328 Figures and 6 Colour Plates. 105s. net. London: Pitman Medical Publishing Co. Ltd. 1958.

Text-book of Surgery. Edited by Guy Blackburn, M.B.E., M.Chir., F.R.C.S. and Rex Lawrie, M.D., M.S., F.R.C.S., M.R.C.P. Pp. xii+1122. Illustrations. 84s. Oxford: Blackwell Scientific Publications. 1958.

BOOK REVIEWS : BOEKBESPREKINGS

STRABISMUS OPHTHALMIC SYMPOSIUM II

Strabismus Ophthalmic Symposium II. Edited by James H. Allen, M.D. Pp. 552. 251 Illustrations. 136s. St. Louis: The C.V. Mosby Company. 1958.

The New Orleans Academy of Ophthalmology has previously published several valuable symposia which have enriched our Literature and increased our knowledge. The present symposium maintains the standard set by its predecessors. Under the distinguished editorship of James Allen, a number of acknowledged leaders in the field have contributed articles which bring the whole subject of Squint completely up to date. The subject is certainly a difficult one and the theoretical aspect is often very complex and technical. Nevertheless, the practising Ophthalmologist will find much to interest and instruct, particularly in the clinical chapters and the indications for the various operative and non-operative procedures.

The book opens with almost 100 pages on the Anatomy of the extrinsic muscles of the eye. Then 21 chapters follow on various aspects of strabismus. Unanimity of opinion can of necessity not be expected. The unorthodox views on the use of atropin and prisms, for instance, expressed by George Guibor, who recently visited this country, are given at length. The book ends with a round-table discussion with the whole panel. Many may find this the most valuable part of the book since many individual rather than general problems are raised and discussed. This is a book which should be a useful addition to the ophthalmologist's library. It is a handsome and well printed book. L.S.

PRINCIPLES OF GYNAECOLOGY

Principles of Gynaecology. By T. N. A. Jeffcoate, M.D., F.R.C.S. (Edin.), F.R.C.O.G. Pp. viii+669+(27). 436 Figures. 84s. 6d.+2s. 5d. Postage. London: Butterworth & Co. (Publishers) Ltd. South African Office: Butterworth & Co. (Africa) Ltd., P.O. Box 792, Durban. 1957.

This book, written by one of the foremost gynaecologists in Britain today, is intended primiraly for the undergraduate student, but it is expected to carry him beyond the qualifying examination and into the period of his hospital residency and even into general practice. Moreover, as the author mentions in his preface, it is aimed at the student at the top of the class rather than the one at the bottom. Specialists will also find much of interest in this book which differs from the standard text-books because it reflects the author's own personal experience of gynaecological practice.

Prof. Jeffcoate did not attempt to keep to generally accepted views, nor are the differing views of various authorities mentioned, but instead he presents his own conclusions which he feels are as rational as present knowledge allows.

At first the book might seem slightly disappointing since it does not cover the whole field of gynaecology, and a number of less common conditions are unduly stressed, but on getting to know the book better, this in fact, becomes its greatest appeal. Two particularly good and useful chapters in this valuable book are 'Abnormal and excessive haemorrhage from the uterus and vagina,' and 'Sex hormone therapy'. It is certainly a book, not for the shelf, but for the desk of everyone interested in gynaecology.

F.G.G.

EMBRYOLOGY AND PATHOLOGY

The Borderland of Embryology and Pathology. By R. A. Willis, D.Sc., M.D., F.R.C.P. Pp. ix+627+(33). 244 Figures. 101s. 3d.+2s. 3d. London: Butterworth & Co. (Publishers) Ltd. South African Office: Butterworth & Co. (Africa) Ltd., P.O. Box 792, Durban. 1958.

Rupert Willis has written another 'best seller'. This book admirably proves his contention that the experimentalist and the histopathologist are mutually dependent and complementary. He has firmly wedded the embryologist, the pathologist, the experimental zoologist and the clinician to one another in a masterly review of a tremendously wide field which is searchlighted by his extensive experience.

Almost 20% of the book deals with normal development and he particularly focuses attention on the histology and functions of the various tissues and organs of the embryo and foetus at different ages. The first 3 chapters are a *must* for every candidate for the Primary Fellowship because a tremendous amount of information scattered throughout the literature is brought together in a simple way which inspires and excites attention. The chapter on gross malformations depicts a dynamic instead of the 'museum' approach to developmental anomalies.

The author has selected malformations of certain organs and tissues which illustrate principles in the mode of genesis, the interdependence of parts, important associations, recognized factors in causation, etc. Of great value are the extensive lists of selected references at the end of each chapter.

In a book which is itself a landmark, it is difficult to choose the most interesting sections—all are vital and thought-provoking, and if there are minor errors they were certainly ignored and unnoticed by the reviewer in his first reading because of the vivid interest of each chapter and the desire (as in reading a good 'thriller') to get on with the next chapter.

Genetic aspects are dealt with and, in parts, comparative (experimental) anatomy is delightfully interlocked with pathological processes in the human e.g. in the chapter on regeneration and repair. The experimental approach is further enhanced by the chapter on the transplantation and culture of tissues, while the appendix presents suggestions for reseach on each of the chapters dealt with. Let it not be said that a postgraduate student must struggle to find a title for a thesis—there are more than a hundred ideas here!

This book has one gross defect—it is too short! The reviewer feels that he would have liked more to each page, but that is how it should be.

Anyone who thinks in terms of embryology and pathology will find this book not only an inspiration but a great timesaving device for it reviews the entire field between the horizons of both subjects.

R.S.

LESIONS OF THE STOMACH

Diffuse Lesions of the Stomach. An Account with Special Reference to the Value of Gastric Biopsy. By Ian J. Wood, M.D., F.R.C.P., F.R.A.C.P. and Leon I. Taft, M.B., B.S., B.Sc. Pp. x+86. 35 Figures. 24s. London: Edward Arnold (Publishers) Ltd. 1958.

This informative monograph presents the results of 10 years of energetic research carried out by the gastroenterology team at the Walter and Eliza Hall Institute and Royal Melbourne Hospital. The title refers to clinical and pathological observations on acute and chronic gastritis, gastric atrophy and pernicious anaemia, and the rare condition of diffuse giant hypertrophic gastritis.

A most valuable first chapter outlines the standard methods of investigation adopted by the authors in cases of flatulent dyspepsia not apparently due to ulcer, cancer, gallstones or pancreatitis, and also in patients with suspected pernicious anaemia or sub-acute cord-degeneration. Test-meal techniques, radiology and gastroscopy are described and their usefulness assessed; but the authors' major contribution is their account of gastric biopsy with the flexible biopsy tube—devised in their unit—and its applications to clinical medicine. The results of this relatively safe and painless procedure, performed successfully 1,736 times on 1,046 patients, throw new light on the diagnosis of chronic dyspepsia, the recognition as a definite entity of active chronic gastritis, the elucidation of haematological problems, the understanding of achlorhydria, and the inter-relationships among gastric lesions.

This slender volume, full of interesting new facts attractively presented, will prove stimulating to both clinicians and investigators.

R.S.M.

EXAMINATION OF THE NERVOUS SYSTEM

Clinical Examination of the Nervous System. 11th Edition. By G. H. Monrad-Krohn, M.D., F.R.C.P. Pp. xx+466. 173 Illustrations. 40s. net. London: H. K. Lewis & Co. Ltd. 1958.

'Good wine needs no bush' and the constant demand and repeated editions and reprints of this book reflects this old saying. The

author maintains his original high standard with each new edition and he keeps good pace with the advances in neurology, though this must inevitably enlarge the book somewhat. The student will find here an unrivalled guide to the systematic examination of the nervous system and if he learns, with experience, to put it into ready practice, the subject will soon cease to mystify him and he will find himself working knowledgeably in this field.

S.B.

CLINICAL OBSTETRICS AND GYNECOLOGY

Clinical Obstetrics and Gynecology. Vol. 1, No. 1. March 1958. This is the first number of a quarterly book series. Pp. 288. Illustrations. $18.000 for four consecutive numbers. New York: Paul B. Hoeber, Inc. 1958.

Clinical Obstetrics and Gynecology represents a worthwhile attempt to bridge the gap between original articles in medical journals, the findings of which are not by any means always acceptable, and the standard text-books.

People whom the editor regards as the leading American experts of obstetrics and gynaecology are asked to contribute articles on how they view various aspects of their specialty and how they handle their patients in practice.

The first issue is certainly a striking volume. The articles are admirably readable and brief. The print and paper are of the highest standard. Even the busiest practitioner will absorb a mass of valuable information with the very minimum of effort, by glancing through this volume.

The first issue deals with medical problems in pregnancy on the obstetrical side, and the management of endocrine problems on the gynaecological side. Although the majority of the articles are of a high standard and will enevitably be of great interest to obstetricians and gynaecologists there are some glaring omissions. For instance, the chapter on abortions completely omits the new knowledge on the incompetent internal os and its treatment. As this is one of the very few substantial advances in obstetrics in recent years the omission is indeed serious.

Nevertheless, if future issues are of this general high standard one can predict that most practicing obstetricians and gynaecologists are likely to become regular subscribers.

G.P.C.

PSYCHOTHERAPY

Progress in Psychotherapy. Volume III. Techniques of Psychotherapy. Edited by Jules H. Masserman, M.D. and J. L. Moreno, M.D. Pp. x+324. $8.50. New York and London: Grune & Stratton, Inc. 1958.

It would indeed be surprising if a collection of over 30 essays on psychotherapy showed an even admixture of competence and relevance. No such surprise hides in this 3rd volume of the series on *Progress in Psychotherapy*.

The essays are grouped under 5 headings but only the first 2 sections, the historical and the methodological, are of much importance. The remaining contributions are not sufficiently developed to be more than pointers in certain directions.

Of the two historical essays, that by Professor Veith is of most interest and contains some unusual information, particularly the Askalepian inscriptions and his quotations from Soranus. And who of us knew that the man who invented the term psychotherapy was Johannes Reil, of island fame?

Professor Frank introduces the question of brain-washing and muses on Chinese 'thought reform' (a euphemism worthy of Kai Lung), pondering whether permissiveness on the part of the therapist is not the consulting-room equivalent of this cogitative reformation. Dr. Ehrenwald writes a stimulating and erudite essay on doctrinal compliance and likens the physicians's therapeutic activity and 'psi-induction' to Heisenberg's principle of indeterminacy, all of which are notable efforts to bring psychology to terms with quantum physics, even though we may not take it very seriously.

What might have been one of the most interesting essays in this collection is the attempt by Dr. Relich to analyse the reasons behind the success and appeal of the analytical-psychological group of therapists in the United States over the group which he labels 'directive-organic' psychiatrists. Unfortunately all he says in favour of the analysts could be equally well applied to the other group. As Humpty-Dumpty pointed out to Alice, 'a nice knock-down argument' can be used in more ways than one.

One must, however, judge a book such as this on the merits of its best contributions, and on these grounds one can say that the standard of this volume is possibly beyond the standard of its two earlier companions.

J.M.MacG.

SURGERY IN INFANCY

Surgery in Infancy and Childhood. A Handbook for Medical Students and General Practitioners. By Matthew White, M.A., M.B., Ch.B., F.R.F.P.S. (Glas.), F.R.C.S. (Edin.) and Wallace M. Dennison, M.D., F.R.F.P.S. (Glas.), F.R.C.S. (Edin.), F.I.C.S. Pp. xii+444. 266 Figures. 45s. net+2s. 1d. Postage Abroad. Edinburgh and London: E. & S. Livingstone Ltd. 1958.

The surgery of infancy and childhood has advanced rapidly during the last decade and it has become obvious that the understanding of the surgical problems of the tender months and years is dependent on a sound academic basis. In spite of the enormous advances in anaesthesia, surgical technique, pre- and post-operative management and nursing services, naught would avail if an early diagnosis were not established. In many cases it is necessary only to be aware of a condition to be able to make a diagnosis. It has been shown repeatedly that a delay in diagnosis significantly raises the mortality rate. The general practitioner is the first to see the sick infant and it is his awareness of the probable surgical lesions that makes an early diagnosis possible.

This volume admirably suits the purpose which the authors have set out to achieve. It serves as an introduction to the senior student and is also of great value to the busy general practitioner. While one may not agree entirely with the way in which some controversial problems are presented, the authors must be credited for their practical approach. I have no hesitation in recommending this volume to both the general practitioner and the senior student as a guide to the early diagnosis and consequent early treatment of surgical problems of infancy and childhood.

A.K.

LUPUS NEPHRITIS

Lupus Nephritis. By Robert C. Muerchke, M.S., M.D., Robert M. Kark, F.R.C.P., F.A.C.P., Conrad L. Pirani, M.D. and Victor E. Pollak, M.B., M.R.C.P. Pp. 145. 43 Figures. 24s. net. London: Baillière, Tindall and Cox Ltd. 1958.

Clinicians in recent years have fully realized the grave prognostic significance of renal involvement in disseminated lupus erythematosus (D.L.E.). The title of this monograph *Lupus Nephritis* may offend the purist, but is has at least the virtue of focusing attention on the kidney.

The study is based on the chemical and pathological investigation of 33 patients with D.L.E. who have been followed up for periods of up to 2 years. The use of percutaneous renal biopsy has proved valuable in gaining an insight into the early renal lesions of D.L.E. although the influence of prior steroid therapy should have been more seriously considered since no less than 22 of the 33 patients had received corticotrophin or cortisone before the first biopsy was done. Of the 10 patients who died, 6 died of severe renal failure.

The morphological findings obtained by repeated renal biopsy and at autopsy are fully described and the illustrations are selected to depict various aspects. The morphological findings were correlated with the clinical tests of renal function. There was good correlation between the grade of kidney damage and the degree of proteinuria and the degree of renal functional impairment.

The difficulties of differentiation from chronic glomerulonephritis on morphological grounds may be considerable. Case 31 seems to illustrate the importance of early renal biopsy in helping to establish the diagnosis of D.L.E.; at autopsy the features were those of chronic glomerulonephritis. Dr. Muerchke *et al*, would no doubt be the first to admit the difficulties in differentiating at least some of the cases, since the renal lesion may be the first recognizable manifestation of D.L.E.

This is a valuable reference book for all those interested in renal disease. The book is well produced and profusely illustrated with good microphotographs.

L.E.

CORRESPONDENCE : BRIEWERUBRIEK

SOUTHERN TRANSVAAL BRANCH: DONATION TO BENEVOLENT FUND

To the Editor: It is customary to acknowledge in the *Journal* donations made to the Benevolent Fund of the Association but I think it is only right that the recent donation made by the Southern Transvaal Branch should receive special mention.

To have raised the unprecedented sum of £2,500 as the result of the holding of social functions is a magnificent achievement. The grateful thanks of my Committee is due to our colleagues in Johannesburg and especially to Mrs. Wolfowitz, wife of the Branch President, and her lady helpers for their splendid effort.

Alan W. Sichel
Chairman, Management Committee of the Benevolent Fund

National Mutual Chambers
Church Square
Cape Town
22 December 1958

MEDICAL COUNCIL ELECTION

To the Editor: May I crave a little space in your correspondence column to thank all my colleagues who gave me their support in the recent election of the South African Medical and Dental Council.

I appreciate very much the honour that has been conferred on me and realize the responsibilities of my membership of the Council. Need I say that I shall try to justify the confidence that my friends have shown in me.

Alan W. Sichel

National Mutual Chambers
Church Square
Cape Town
22 December 1958

MEDICAL COUNCIL ELECTION

To the Editor: May I through the medium of your *Journal* express my sincere appreciation to the members of the profession who, once again, have honoured me by appointing me as one of their representatives on the Medical Council.

It is an honour that I deeply appreciate.

R. Lance Impey

Finchcroft
Talana Road
Claremont, Cape
22 December 1958

NATIONAL INSURANCE AND MENTAL HEALTH

To the Editor: His Honour the Administrator of Natal, Mr. A. E. Trollip, stressed, at the annual dinner of the Natal Coastal Branch of the Medical Association of South Africa, held in Durban last month, the necessity for the introduction of some form of National Health Insurance to meet the needs of the 'middle income group'.

If the Government intends to implement such a scheme it is essential that we should make certain that our patients are fully covered for mental illnesses. At present many accident and sickness policies, issued by insurance companies, contain restrictive clauses; the most common clause excludes liability for 'mental disorder'. For example, a patient was referred to me by his doctor with a diagnosis of 'nervous indigestion' which had developed as a result of pressure of work and shortage of staff in his printing business. This situation had necessitated his working exceedingly long hours over a considerable period of time. On investigation he was found to be suffering from a stress reaction associated with acute anxiety and depression. Sick-leave was recommended but the insurance company, on receipt of his claim, took legal advice and refused to accept liability on the grounds that the patient was suffering from a 'mental disorder'.

Now it has been shown that about ⅓rd of all patients attending the outpatient's departments of general hospitals are psychiatric cases and another ⅓rd suffer from psychosomatic disorders. This means that over 60% of illness is not legally covered by many of the present insurance policies.

Further, government departments and provincial and municipal administrations have also failed to appreciate that it is just as much an illness when the behaviour of the whole organism is affected as when a single organ of the body is behaving abnormally. In consequence their employees suffer great hardships in not being compensated for mental illnesses.

The 2nd Report of the World Health Organization Expert Committee on Mental Health emphasized the indispensability of mental activities in public health practices. Buckle said that as we see the field of mental illness today in the more culturally and economically developed countries, we recognize that there is an insanity rate of about 2% amongst the people of these countries, and a neurosis rate—defined at the level of partially disabling illness—of about 10%. It is becoming evident that medicine must revert once again to the treatment of persons, not merely the treatment of diseases. Treatment is no longer the affair of numerous specialists, isolated from one another. The public health services exist to prevent and modify illness and must assume responsibility for the total well-being of their patients.

Coleman pointed out that any approach to illness which does not consider psychological and social data is not only likely to be unrewarding but is also in a sense reducing the practice of medicine from a profession to a technical level. I think it worth re-emphasizing that man is the proper study of mankind, even in his illness, and that the study of man is incomplete without a knowledge of the inner and outer forces which determine his relation to himself and to his fellow men.

Peterson emphasized that mental health programmes lag far behind public health (or physical disease) programmes. He said that if the incidence of physical disease reached the proportion of many of our social ills with mental and emotional causes (alcoholism, delinquency, suicide and even divorce), not to mention classical mental disease, an epidemic state would be declared and strong measures would be taken to combat it.

Today medicine is interested in the human being as one integrated inseparable whole and it is our duty as doctors to insist that the whole man is insured.

B. Crowhurst Archer

619 Sanlam Buildings
Durban
15 December 1958

EXPLOITATION BY LOCUMS

To the Editor: The letter 'Exploitation by Locums' by 'Noel'[1] which was published in the *Journal* of 13 December 1958 is of interest to all of us, particularly to general practitioners.

There are 2 other aspects of the problem to be considered: the first is that most locums, particularly those who are recently qualified, *need* the money to repay various loans which have been necessary to help them along the 6 years of study, not to mention money they have needed to live and clothe themselves. Some have even had families to support.

The second aspect of the problem is also the solution, and here I take a shot in the dark regarding 'Noel'. If he is to be classed as an average general practitioner among us, I would say that he could probably pay a locum £200 and more a month for a whole year from his outstanding debts alone. Let us face the facts: our profession is usually the last to be paid and often the least considered. How often hasn't one heard the saying 'The doctor is rich, he can wait'? If incurring bad debts were made a criminal offence instead of a civil offence, we would all get our due and the problem of fees for the locum would be solved.

Just how serious this bad-debt problem is, may be judged from an article in the *Sunday Times* of 14 December 1958 where half of a deceased doctor's estate of £35,000 was considered bad debt.

19 December 1958 *Young, and grey already*

1. *Noel* (1958): S. Afr. Med. J., 32, 1204.